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VOLUME 9 IN TWO PARTS—PART I NUMBER 3

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# A CLINICAL STUDY OF DISEASES OF THE CIRCULATION OF THE EXTREMITIES: A DESCRIPTION OF A NEW METHOD OF EXAMINATION *

BARNEY BROOKS, M.D.

AND

FRED A. JOSTES, M.D.

ST. LOUIS

There is, at the present time, no accurate method of estimating the extent of diminution in the circulation of the extremities, resulting from disease of the blood vessels. Furthermore, it is often impossible, by methods of examination now used, to determine whether or not actual circulatory disturbance exists. The absence of any of the clinical manifestations of circulatory disease, when a good pulse is present in all the large arteries and the color of the skin is normal, may be taken as conclusive evidence of normal circulation. The presence of gangrene or of even a marked blanching of the skin of an extremity is undisputed evidence of a deficient circulation of blood. Between these two extremes, however, are found the greatest number of instances of disease of the blood vascular apparatus, and, in these instances, it is impossible to estimate with accuracy the degree of diminution of circulation, and often impossible to determine whether any really exists. For example, a man aged 55, who comes complaining of pain in the legs may show none of the gross manifestations of arterial disease and whether the pain is the result of bad posture or of bad circulation cannot be determined. If gangrene, the grossest manifestation of deficient circulation, exists, the degree of disturbance of the circulation in the adjacent tissues is a matter of conjecture. This is of great importance in determining the advisability of amputation and the selection of its level.

Moszkowicz [1] described the well known skin hyperemia test, in which the extent of hyperemia following a period of anemia is observed, the development of a visible hyperemia being considered as evidence of a good circulation, and the development of a visible differentiation between

* From the Department of Surgery of Washington University, St. Louis.

1. Moszkowicz: Mitt. a. d. Grenzgeb. d. Med. u. Chir. **12**:216, 1907.

hyperemia and anemia as a sign of value in the choice of the level of amputation for gangrene. Buerger has made use of the visible change in the blood content of the extremity at different angles of elevation in order to estimate the degree of circulatory disease. Both of these clinical methods are valuable aids in the diagnosis and treatment of obstructive disease. Both have the disadvantage of being dependent on indefinite color changes.

The only exact method of determining the efficiency of the circulation in an extremity would be one by which the actual amount of blood flowing through the tissues could be measured. Stewart [2] has described a method by means of which a volume flow can be determined, which can not be less than the actual flow. The preliminary experiments of this work were carried out with a calorimeter and the method was similar to that employed by Stewart. It was found, however, that the limits of error were so great that the method was valueless when applied to the study of the human extremity in which the volume flow of blood was diminished. The immersion of an extremity in a fluid, the temperature of which is different from that of the extremity, results in an alteration of the circulation. The temperature of the tissues of the distal portion of an extremity also fluctuates through a wide range in relatively brief intervals.

The method of study used in this paper is one by which the temperature of the tissues and the changes in temperature of the tissues have been measured. The temperature has been measured by the use of the thermocouple galvanometer method.

The thermocouple and galvanometer were used as long ago as 1835, by Becquerel and Breschet [3] to determine the temperature of different tissues in different diseases. Lombard,[4] Rosetti,[5] Hill [6] and others have used the thermocouple and galvanometer to measure the temperature of tissues in various physiologic investigations. Macleod [7] used a thermocouple mounted in a hypodermic needle to measure the temperature of the tissues after the application of heat and cold to the body surface. Crile, Rowland, and Hosmer [8] have used the thermocouple for the measurement of the temperature of the liver and brain in various physiologic

2. Stewart: Am. J. Physiol. **27**:20, 1910.
3. Becquerel and Breschet: Comp. Rend. **1**:28, 1835.
4. Lombard: New York M. J. **5**:98, 1867.
5. Rosetti: Annal. d. Chini. et d. Phys., Series 4 **13**:68, 1868.
6. Hill: J. Physiol. **43**:433, 1907.
7. Macleod, J. J. R.; Self, A. R., and Taylor, N. B.: Lancet **2**:645 (Sept. 25) 1920.
8. Hosmer, H. R.; Rowland, A. F., and Crile, G. W.: Am. J. Physiol. **62**: 341-382 (Oct.) 1922.

and pharmacologic experiments on animals. Mudd and Grant [9] used this method in the study of temperature variations of the mucous membranes.

The apparatus used in this study consisted of a thermocouple of iron and constantan, a Leeds and Northrup portable galvanometer No. 24 B, and a thermos bottle. The apparatus is illustrated in Figure 1. The thermocouple was constructed by introducing an insulated constantan wire into a long thin needle. The wire was coiled and soldered at the point, sealing the end of the needle. The sleeve of the needle, being an alloy, was excluded from the system by soldering the iron wire to the shaft of the needle. A gutta percha handle was attached to the needle (Fig. 2). The needle was sterilized by immersion in phenol (carbolic acid). All wires were insulated and contacts other than that of the tip of the needle were protected from temperature changes as much as possible.

The constant temperature point was obtained by immersion of the appropriately protected iron constantan junction in a thermos bottle containing a thermometer and stirrer. The temperature in the thermos bottle was maintained at 30 C. by the occasional addition of a small quantity of hot water.

The galvanometer was interposed in the iron wire. The galvanometer was calibrated by placing the needle in a second thermos bottle containing a thermometer and water at 35 C., and reading the galvanometer at each one-tenth degree fall in the temperature of the water. The calibration was repeated several times, and so little variation in the value of the galvanometer readings was found that correction has not yet been necessary. The rapidity of change in the temperature of the tissues in this study made it unnecessary to read the temperature to a greater degree of accuracy than one tenth of a degree centigrade.

According to the method of procedure, the patient to be examined was placed in bed in a closed room, the temperature of which was recorded. Both extremities to be examined were uncovered for a period of one-half hour before the examination was begun. The site at which the tissue temperature was to be taken was prepared with tincture of iodin and alcohol. A small area of skin was infiltrated with one drop of 0.5 per cent. procain. The thermocouple needle was then introduced into the tissues and the galvanometer reading and the time recorded. The site chosen in most instances was the great toe. The needle was inserted to a depth of 6 mm., into the subcutaneous fat pad. The procedure is almost painless.

The temperature of the tissue was taken at intervals of from one to five minutes, according to the rapidity of change. There was in almost

9. Grant, S. B.: J. M. Res. **40**:53 (May) 1919.

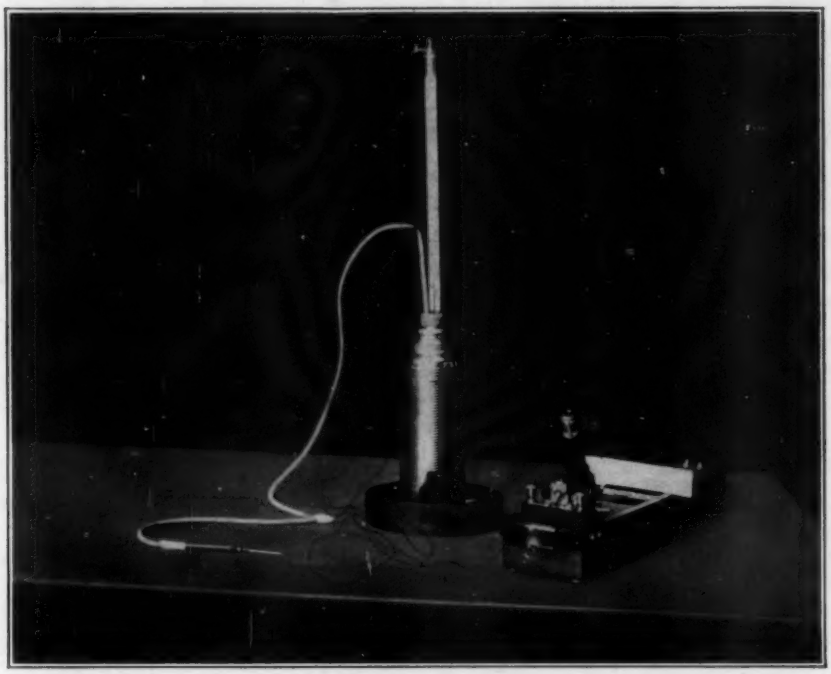

Fig. 1.—The apparatus used; the "thermocouple" needle, the galvanometer, and the thermos bottle for maintaining a constant temperature of one contact of the thermocouple.

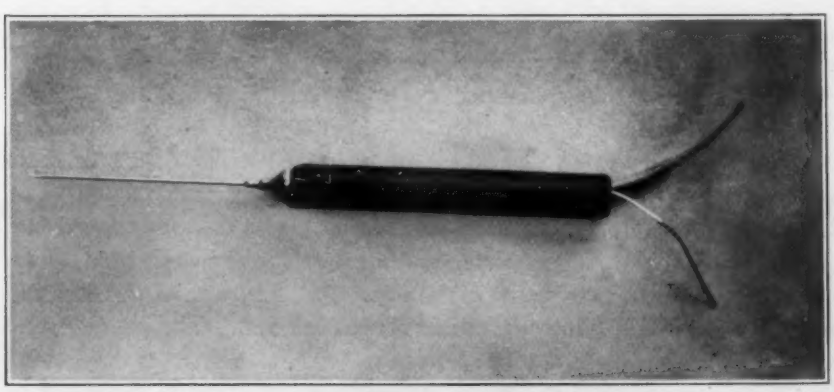

Fig. 2.—The "thermocouple" needle.

every instance a rise in temperature of the tissues immediately after the insertion of the needle. This was not due to absorption or conduction of heat by the needle, because similar galvanometer variations did not take place if the needle was inserted into water at the same temperature as the tissues. After the preliminary rise, the temperature of the tissue usually became more nearly constant, but if observations were continued over any considerable period, rather marked spontaneous fluctuations were found. At the time when the temperature of the toe was nearly constant, a tourniquet was applied to the thigh tight enough to obstruct the arteries. Following this procedure, the temperature of the toe immediately began to fall. The tourniquet was left in place, if possible, for ten minutes, the temperature being recorded at one minute intervals. The tourniquet was then quickly removed and the temperature readings made at as short intervals as possible. The construction of a graphic method of recording would greatly improve the accuracy of this record. The change in temperature after the removal of the tourniquet varied in different individuals, and the relative time of the appearance of the temperature change after the removal of the tourniquet is the most important observation to be discussed in this paper. If the temperature changes of the tissues resulting from the application and removal of a tourniquet are plotted, the curve obtained is characteristic of the condition of the circulation in the tissue examined.

The results obtained in the following clinical applications of this method of examination illustrate the method of procedure as well as the value of the results obtained in the diagnosis and treatment of circulatory diseases of the extremities.

## REPORT OF CASES

CASE 1.—A white man, aged 28, was admitted to Barnes Hospital, Nov. 26, 1923, for appendectomy for chronic appendicitis. The examination, made ten days after operation, revealed no manifestations of disease. The record of the temperature in the tissues of the great toe before, during, and after the application of a tourniquet to the thigh is shown in Figure 3. The temperature of the toe remained at nearly 34.4 C., for approximately thirty minutes. The tourniquet was applied for ten minutes. The temperature of the toe fell to 31.2 C. The tourniquet was removed, and the temperature of the toe rose in three minutes to 34 C. The rise was accompanied by a visible hyperemia of all of the extremity distal to the site of application of the tourniquet.

CASE 2.—A man, aged 24, was admitted to Barnes Hospital, Nov. 26, 1923, for the repair of an inguinal hernia. There was no evidence of any other disease. The results of measurements of the temperature changes in the deep tissues of the great toe in response to the application and removal of a tourniquet are shown in Figure 4. The temperature of the tissues was only a little above room temperature at the beginning of the examination. There was a slight rise immediately after the introduction of the needle followed by a

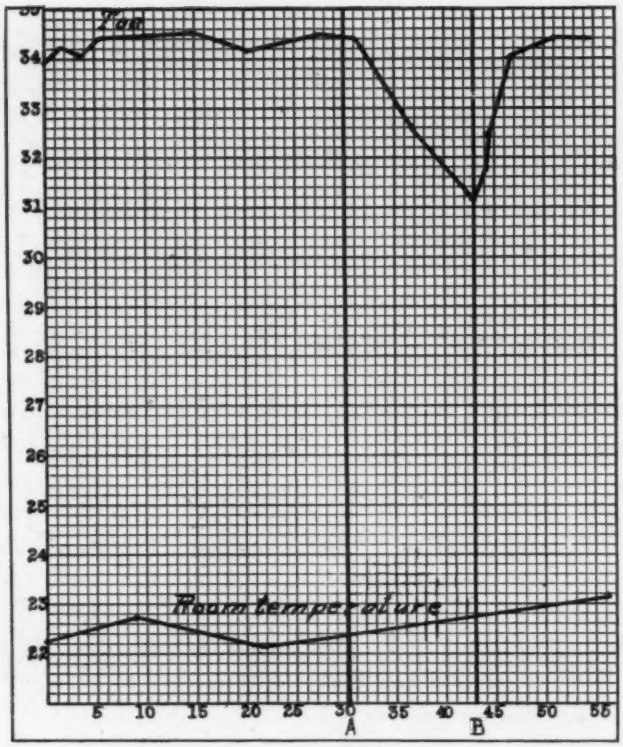


Fig. 3.—Curve obtained in Case 1. Note the fall and rapid recovery of temperature in the deep tissues of great toe as a result of the application and removal of a tourniquet on the thigh of a normal individual. *A*, tourniquet on; *B*, tourniquet off.

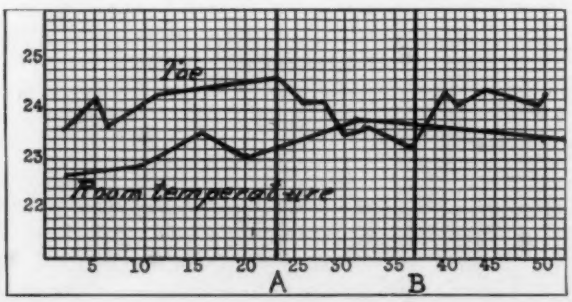


Fig. 4.—Curve obtained in Case 2, at a time when the temperature of the tissues of the toe was low. Note that the temperature, however, rose immediately to its former level after the removal of the tourniquet. Compare with Figure 5. *A*, tourniquet on; *B*, off.

slight fall. Subsequently, a fairly constant level was maintained about one degree centigrade above room temperature. The application of the tourniquet to the thigh was followed by a progressive fall in temperature of the toe to a level below room temperature. The removal of the tourniquet resulted in a rise of temperature which began immediately after the tourniquet was released. Four minutes later, the temperature was approximately the same as at the time the tourniquet was applied.

The same toe was examined two days later under apparently the same conditions, and the results shown in Figure 5 were obtained. In this examination, the temperature of the toe was about 4 C. above room temperature at the beginning of the test, although the foot had been exposed the same length of

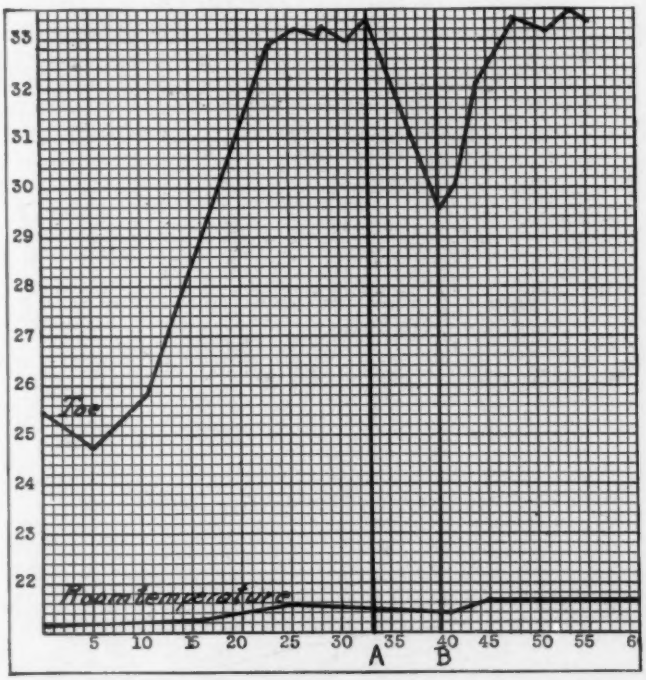


Fig. 5.—Curve obtained in Case 2, at a time when the temperature of the tissues of the toe was relatively high. Note the rapid fall of temperature on application of the tourniquet to the thigh, and the immediate rise to its former level after removal of the tourniquet. Compare with Figure 4. These curves are considered indicative of normal blood vessels. *A*, tourniquet on; *B*, off.

time as in the first examination. The temperature then rose during the following twenty-five minutes to approximately 33 C., remaining at that level until the tourniquet was applied. The application of the tourniquet resulted in a marked fall of temperature, and its removal in a sudden rise.

The record obtained in Case 1 is similar to those obtained in most instances in which there was presumably a normal circulation. The temperature of the distal tissues was considerably above room tempera-

ture after exposure of one-half hour. The application of a tourniquet is followed by a fall in temperature of the tissue distal to the tourniquet. The removal of the tourniquet is immediately followed by a rise in temperature of the tissues, which reaches approximately the original level in from two to seven minutes.

The results obtained in Case 2 illustrate the fact that the temperature of the distal tissues of an extremity varies widely without the existence of circulatory disease. The temperature of the deeper tissues of the toe of a normal individual may closely approximate room temperature at one time and closely approximate body temperature at another. The changes in temperature following the application and removal of the tourniquet in either instance, however, are similar. The

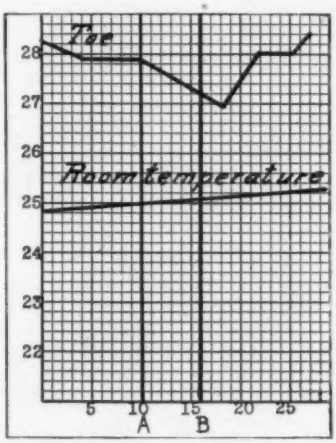


Fig. 6.—Curve obtained in Case 3, in which there was arterial disease associated with diabetes. Note that the fall in temperature resulting from the application of the tourniquet continued for two minutes after the tourniquet was removed. *A*, tourniquet on; *B*, off.

fall in temperature due to the application of a tourniquet is followed by an immediate and rapid rise after its removal.

Case 3.—A white man, aged 50, was admitted to Barnes Hospital, Dec. 26, 1923. Five weeks before admission, the small toe of the right foot had been injured when a pocket knife dropped on it. The toe had become discolored, and severe pain had developed in the foot and leg. Examination showed the patient to have diabetes. There was gangrene in the fourth and fifth toes and in a portion of the dorsum of the foot.

The temperature changes in the deep tissues of the great toe resulting from the application to and the removal of a tourniquet to the thigh are illustrated in Figure 6. The pain in the foot and leg was so severe that the patient could not endure the tourniquet for a longer period than six minutes. On the application of the tourniquet, the temperature of the toe began to fall. On its removal, there was not an immediate rise of temperature in the toe. On the

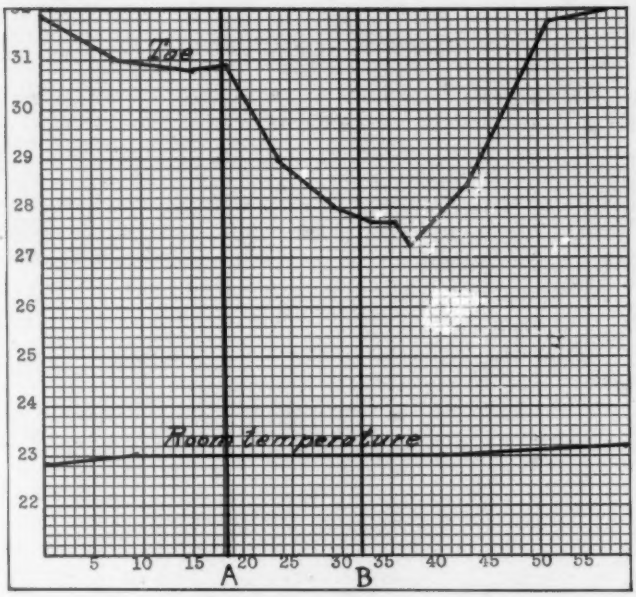


Fig. 7.—Curve obtained in Case 4. Left foot, showing that there is an interval of five minutes between the removal of the tourniquet and the beginning of the rise in temperature of the tissues of the toe. *A*, tourniquet on; *B*, off.

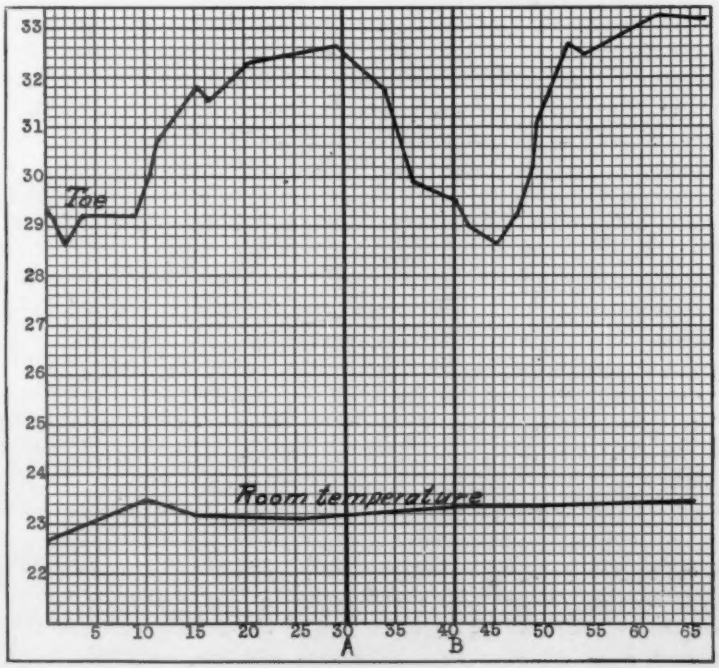


Fig. 8.—Curve obtained in Case 4. Right foot. Note that the curve is similar to that obtained in the left foot. The temperature of the toe did not begin to rise until five minutes after the tourniquet was removed from the thigh. *A*, tourniquet on; *B*, off.

other hand, it continued to fall for a period of three minutes, after which the temperature rose in a period of five minutes to its previous level.

Subsequently, the arteries of the extremity were injected with a solution of sodium iodid, and roentgenograms showed extensive arteriosclerosis of all the large arteries of the leg. The arterial disease was such that a thigh amputation seemed advisable. Examination of the amputated specimen showed almost complete obliteration of both the posterior and anterior tibial arteries. The peroneal artery was partially obstructed by atheromatous patches.

Case 4.—A white man, aged 52, was admitted to Barnes Hospital, Nov. 28, 1923. For eight years previous to admission the patient had suffered with pains in the legs. The pain was always brought on or made worse by walking. The symptoms had been most severe in the left leg. The patient had noticed that the feet became very pale when the pain was most severe. At the time of admission, he was unable to walk more than the length of a city block without resting. The clinical picture was characteristic of "intermittent claudication." Examination showed both feet cyanotic in a dependent position, and blanched if elevated. There was no palpable pulse in either foot or popliteal space. If the patient walked the feet became cadaverous in appearance. When he lay in bed, touching the plantar surface of either foot caused all the muscles of the lower extremities to become rigid. This rigidity could be made to disappear only by passive flexion of either knee. With the development of the spastic contraction of the muscles of the legs, there was a marked blanching of both feet. The history and the physical signs simulated the condition frequently described as Raynaud's disease.

The temperature changes in the deep tissues of the left great toe in response to the application and removal of a tourniquet on the thigh are shown in Figure 7. The results of a similar examination of the right great toe are shown in Figure 8. These curves show, on application of the tourniquet, a fall in temperature continuing for a period of five minutes after the removal of the tourniquet.

Afterwards, the left femoral artery was explored by a surgical operation. The artery did not pulsate. The walls of the vessel were so thickened that the vessel could not be compressed. The operation of Leriche's periarterial sympathectomy was performed. As recorded two days after this operation, the temperature changes in the left great toe in response to the application and removal of a tourniquet on the thigh are illustrated in Figure 9. The result obtained was similar to that obtained before operation. A period of seven minutes elapsed between the time the tourniquet was removed and the beginning rise in the temperature of the toe. There were no other objective signs of change in the condition as a result of the operation of periarterial sympathectomy.

Case 5.— A white man, aged 72, was admitted to the Barnes Hospital, Jan. 3, 1923, suffering severe pain and ulceration of the right foot. The pain and ulceration had been present for about three months. There were indefinite symptoms of circulation deficiency during the last seven years.

Examination showed the typical clinical manifestations of the marked arteriosclerosis of advanced age. The right foot was extremely painful. It was cyanotic in the dependent position. It became cadaverous if elevated. There were small spots of gangrene in the skin of the toes. No pulse could be felt in any of the arteries of the foot or leg. The blood pressure in the

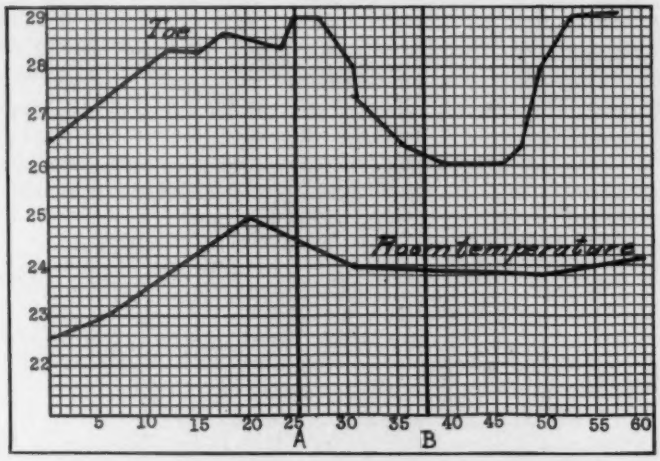


Fig. 9.—Curve obtained in Case 4. Left foot. Two days after periarterial sympathectomy of the left femoral artery. Compare with Figure 7. *A*, tourniquet on; *B*, off.

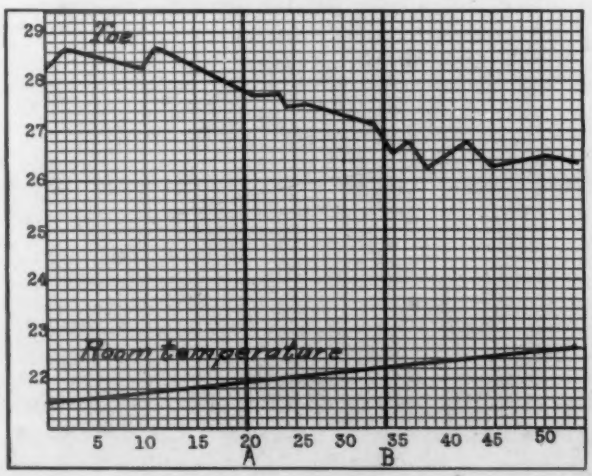


Fig. 10.—Curve obtained in Case 5. Right foot. Note that no influence is produced on the temperature of the toe by either the application or the removal of the tourniquet. *A*, tourniquet on; *B*, off.

brachial artery was: systolic 230, diastolic 90. Roentgen-ray examination showed extensive calcification of all the large arteries of the lower extremities.

The temperature changes in the right great toe in response to the application and removal of the tourniquet on the thigh are shown in Figure 10. The results of examination of the left extremity are shown in Figure 11. Neither the application nor the removal of the tourniquet caused any temperature changes in the right toe. The temperature was falling at the time the needle was introduced. The rapidity of fall was not decreased by the application of the tourniquet. The fall in temperature continued after the removal of the tourniquet, except for a slight temporary rise appearing seven minutes after the tourniquet was off.

Subsequently, the femoral artery was exposed by surgical operation. The vessel was markedly thickened and calcified. The artery was injected with

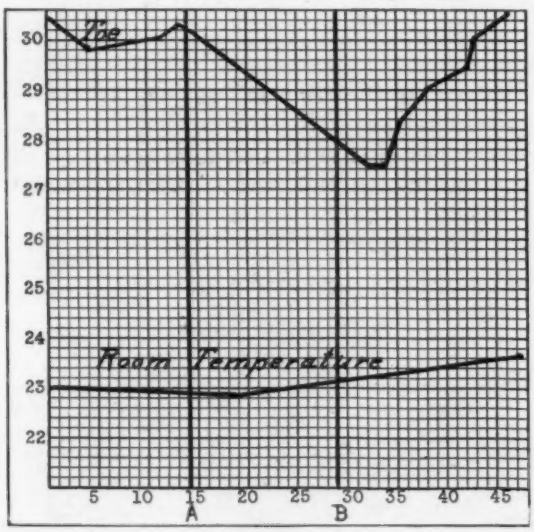


Fig. 11.—Curve obtained in Case 5. Left foot. Note that the temperature of the toe did not begin to rise until 4.5 minutes after the removal of the tourniquet. *A*, tourniquet on; *B*, off.

sodium iodid and the roentgen-ray photographs showed almost complete obliteration of the anterior and posterior tibial arteries. The lumen of the peroneal artery was partially obstructed by atheromatous patches (Fig. 12).

The temperature changes in the tissues resulting from the application and removal of a tourniquet were similar in Cases 3, 4 and 5. All these cases were instances of known disease of the arteries of the extremities. In Cases 3 and 4, the temperature of the tissues fell as a result of the application of a tourniquet. There was a rise of temperature after the tourniquet was removed, but this rise did not begin until from three to seven minutes after the removal of the tourniquet during which interval the temperature continued to fall. In Case 5, in which there were

marked clinical manifestations of arterial obstruction confirmed by intra-arterial injection of sodium iodid and roentgen-ray examination, neither the application nor the removal of the tourniquet resulted in temperature changes in the distal tissues.

CASE 6.—A white man, aged 54, was admitted to Barnes Hospital, Oct. 28, 1923, for severe diarrhea, and swelling and discoloration of both feet. Previous

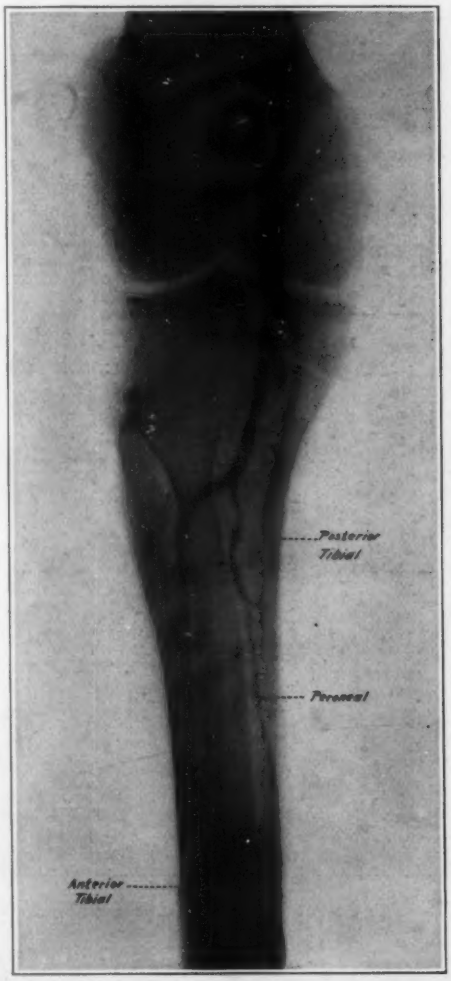


Fig. 12.—Roentgenogram of the arteries of the right leg (Case 5), showing complete absence of lumen in the distal portions of both the anterior and posterior tibial arteries. The peroneal artery shows extensive arteriosclerosis.

to his admission, the patient had been in an outdoor camp, and the shoes and stockings had been worn continuously for a period of one week. On removal of the shoes and stockings discoloration of the toes of the right foot had been noticed. Examination revealed definite gangrene present in all the toes of the right foot and in the nail bed of the great toe on the left foot. Both feet were

swollen and red. No pulse could be felt in the right foot. There was a pulse in the left dorsalis pedis artery. The extent of gangrene in the right foot is illustrated in Figure 13.

The temperature reactions in the tissues immediately adjacent to the line of demarcation of gangrene in the right foot are shown in Figure 14. The temperature reactions in the tissue of the left great toe are illustrated in Figure 15. The observations obtained in each instance were similar to those obtained in tissues of known normal circulation.

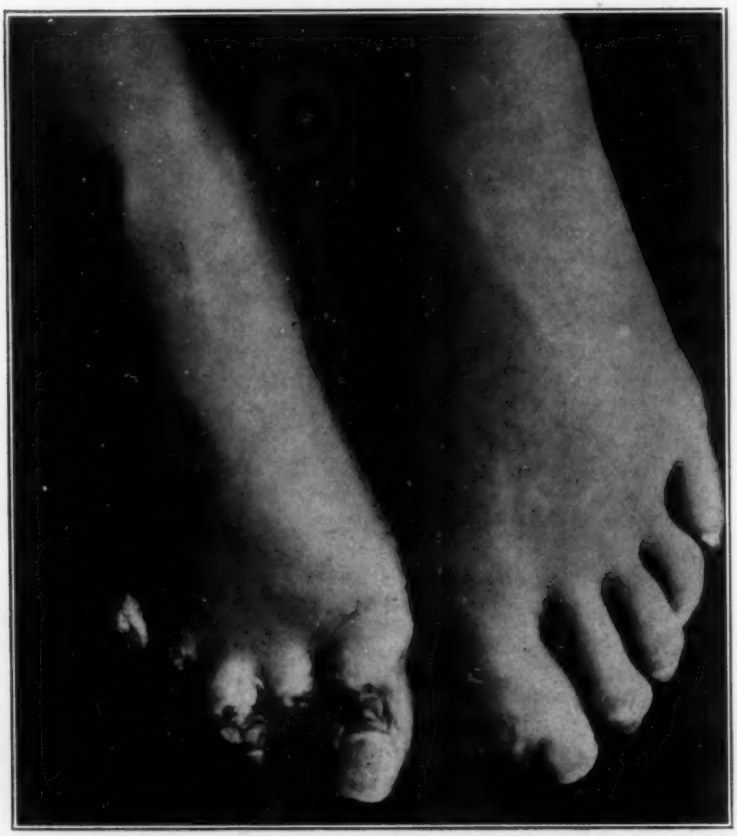

Fig. 13.—The extent of gangrene present in Case 6.

The examinations were repeated several times during the patient's stay in the hospital, and the results obtained were always the same. The gangrenous toes of the right foot spontaneously separated and the wounds healed by granulation. At the time of the patient's discharge from the hospital there was no evidence of disease of the circulation of the extremity, and it was our opinion that the gangrene was caused by continuous local anemia resulting from the long continued exposure to cold and the wearing of tight shoes, especially during periods of inactivity.

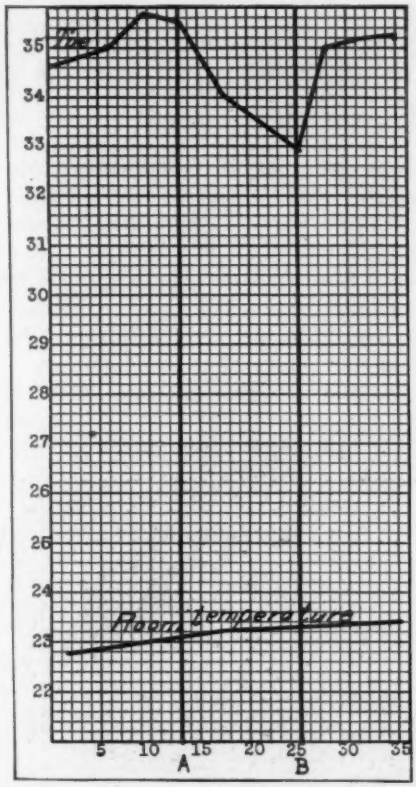


Fig. 14.—Curve obtained in Case 6. Near the line of demarcation of gangrene. Note that the temperature rises immediately after the removal of the tourniquet from the thigh. *A*, tourniquet on; *B*, off.

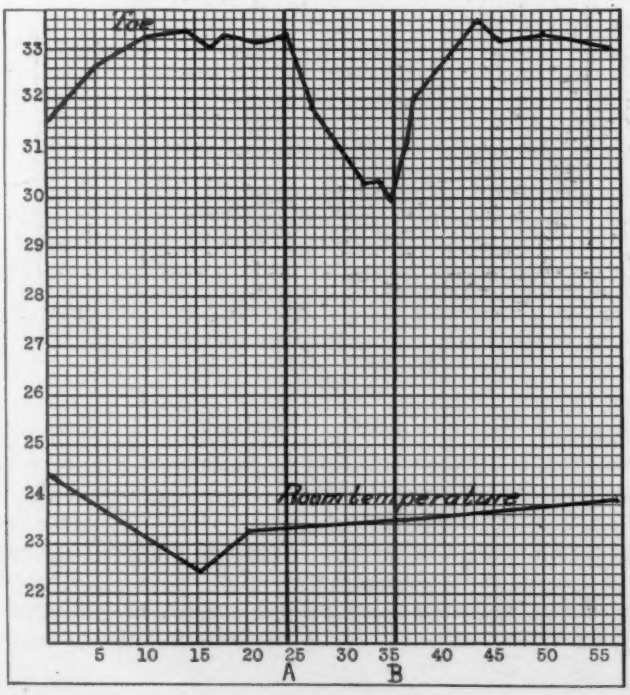


Fig. 15.—Curve obtained in Case 6. Left great toe. Note that the curve is similar to that obtained in normal individuals. There was an area of gangrene in the toe. *A*, tourniquet on; *B*, off.

Case 7.—A white man, aged 53, was admitted to Barnes Hospital, Nov. 20, 1923, for the treatment of diabetes and chronic pulmonary tuberculosis. One day after admission, the patient developed extensive blebs on both feet and legs; these developed into ulcers. It was believed that the probable cause of the ulceration was deficient circulation from arterial disease, so frequently associated with diabetes.

The temperature reactions in the right great toe are shown in Figure 16. The result obtained in the left great toe was similar. In each instance the temperature, which had fallen as a result of the application of the tourniquet to the thigh, rose after its removal. There was an interval of approximately

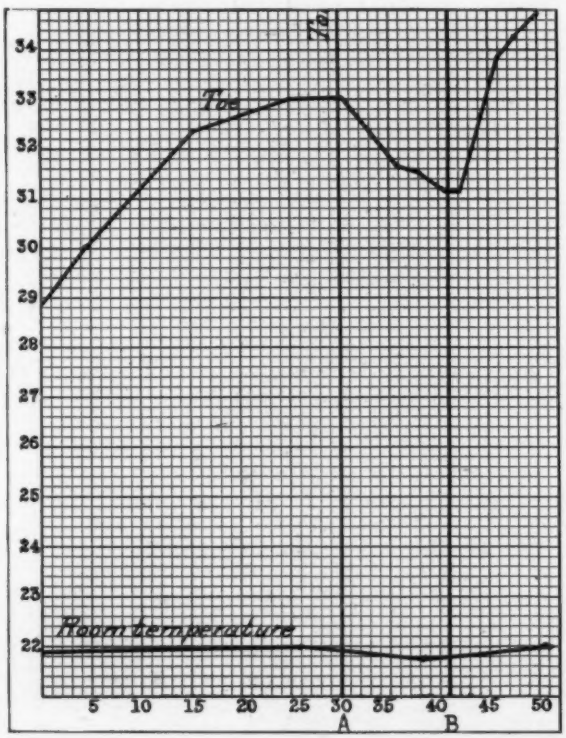


Fig. 16.—Curve obtained in Case 7. Right foot. Note that the curve is similar to that obtained in normal individuals. *A*, tourniquet on; *B*, off.

one minute between the removal of the tourniquet and the beginning of the temperature rise.

Subsequently, the right femoral artery was exposed by surgical operation and injected with sodium iodid solution. The roentgenogram of the injected arteries is shown in Figure 17. All the major arteries of the leg are injected. All arteries appear smaller than normal. There is a slight constriction of the lumen of the anterior tibial vessel at its origin.

The temperature reactions obtained and the demonstration of the preservation of the lumen of all of the major arteries indicated that the ulceration was

not due to disease of the circulation. Subsequent examination showed marked diminution in the pain sense of the lower extremities, and the patient recalled having stood in a tub of hot water before realizing it was "too hot."

Case 8.—A white man, aged 44, was admitted to Barnes Hospital, Jan. 1, 1924, for the treatment of a large epithelioma of the distal third of the leg. An ulcer had been present for nineteen years. During the last eleven months, the

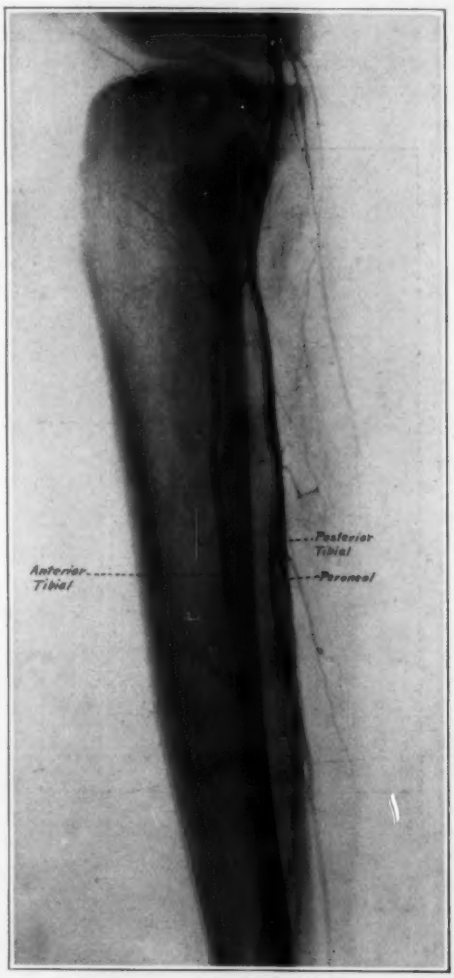


Fig. 17.—Roentgenogram obtained from intra-arterial injection of sodium iodid in Case 7. All the major arteries are nearly normal.

ulceration had changed in appearance and had become more extensive. A piece had been removed and examined. The examination showed the typical appearance of epithelioma arising in a chronic ulcer.

Examination revealed a large ulcerating epithelioma on the medial surface of the distal third of the left leg. The foot was swollen and edematous. No

pulse could be felt. There were no visible varicose veins. The Wassermann test was negative. The etiology of the ulcer was not clear. Amputation was indicated, and the site of amputation was a matter to be determined.

The temperature reactions in the left great toe are illustrated in Figure 18. There was a fall of temperature beginning with the application of the tourniquet. The removal of the tourniquet was followed by an immediate and rapid rise. The femoral artery was exposed by surgical operation. The vessel appeared normal. It was injected with sodium iodid. A roentgenogram of the injected vessels showed that all the major arteries of the extremity were apparently normal, and this normal condition of the arteries of the leg was confirmed by examination of the amputated specimen.

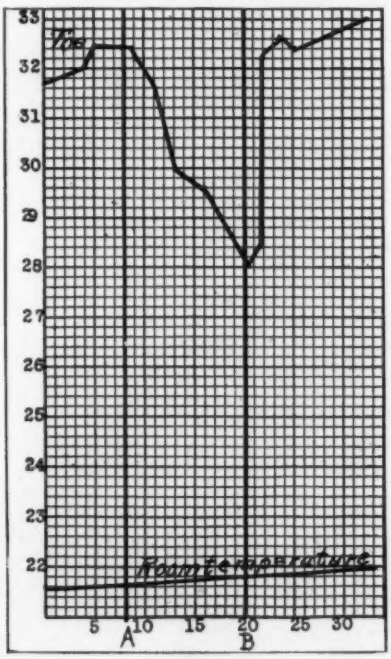


Fig. 18.—Curve obtained in Case 8. It is similar to that obtained in a normal individual. *A*, tourniquet on; *B*, off.

## COMMENT

From the results obtained in thirty-seven examinations of eighteen individuals it would seem that the study of the temperature changes in the tissues resulting from alternate obstruction and release of the obstruction of the arterial circulation gives valuable information as to the presence or absence of arterial disease. Obviously, the temperature of the distal tissues of a normal extremity will fall if the circulation of blood is completely obstructed, and will rise if the obstruction is removed, assuming the extremity is exposed to ordinary room temperature. In all persons examined, whose arteries were presumably normal,

the temperature of the distal tissues fell during the period the arterial circulation was obstructed and rose *immediately* after the removal of the obstruction to approximately its former level. In all persons examined, whose arteries were known to be obstructed by disease, the temperature of the distal tissues fell during the period the arterial circulation was obstructed, but did not begin to rise immediately after the release of the obstruction. In all instances, the fall in temperature of the distal tissues continued for a period of from two to seven minutes after the removal of the obstruction. In one instance of very marked arterial disease (Case 5), no rise in temperature was observed. The length of time elapsing between the removal of the tourniquet and the beginning of the rise in temperature of the distal tissues seems to be an index of the magnitude of obstruction of the large arteries. Another factor to be considered is the rapidity of the rise of temperature after it has once begun. In all instances of presumably normal circulation, the rise has been rapid, the temperature rising as much as from 1 to 3 degrees C. per minute. In most instances in which the arteries have been known to be diseased the rise has been less rapid. In one instance (Case 4), where disease of the large arteries was known to be present, the rise was relatively rapid following a relatively long period of delay. In another instance (Case 7), where there was no demonstrable disease of the large arteries, the rise was relatively slow, and there was only a very short period of delay. This patient was very much emaciated and was suffering from severe diabetes and advanced pulmonary tuberculosis. It would seem, therefore, as if the rapidity of the rise of temperature might be an index of the condition of the circulation in tissues in a sense independent of the condition of the main arteries of the extremity. In the case of patients receiving repeated examinations, the curves obtained have been similar. Thus, it would seem as if the form of the curve obtained was characteristic of the condition existing. If there is a continued fall of temperature in the distal tissues after the tourniquet is removed, obstruction of the large arteries exists. The significance of the rate of rapidity in the rise in temperature after it has once begun is not now clear. Further study will be necessary to differentiate the significance of these two factors.

# BRODIE'S ABSCESS *

MELVIN S. HENDERSON, M.D.
AND
HAROLD E. SIMON, M.D.
Fellow in Surgery, the Mayo Foundation
ROCHESTER, MINN.

Brodie's abscess is a chronic infection in the cancellous tissue at the extremities of the long bones, manifesting itself as a well localized pyogenic abscess, usually without external fistula or sequestrum. Although it is a distinct pathologic entity with characteristic symptoms and roentgen-ray findings, and a fairly well established etiology, the condition is often not diagnosed. Expansion of the term to include other conditions, such as medullary abscess, sclerosing osteitis, and even tuberculous abscess of the long bones, has been largely responsible for the confusion that exists in the literature as to its exact nature.

## HISTORY AND LITERATURE

In 1830, Brodie rescued this group of cases from the general classification of "white swelling," and, although unaided by the roentgen ray and modern laboratory methods, he gave a clinical description which has not been equaled. He described nine cases.[1] Comparatively little has been written on the subject since then. Thomson's article,[2] in 1906, in which he reported 161 collected cases, is probably the most complete contribution available. Gross,[3] in 1901, collected 141 cases, but this report is not available at this time. Brickner [4] and McWilliams have each reviewed the subject and reported cases. Since 1906, we have

* From the Section on Orthopedic Surgery, Mayo Clinic.

1. Brodie, B.: Lecture on Abscess of the Tibia, London Med. Gaz. **36**:1399-1403, 1845; Pathological and Surgical Observations on the Diseases of the Joints, London, Longman, 1836, 354 pp.; Works of Sir Benjamin Brodie, London, **2**:311 and **3**:403 (Quoted by Ogilvie, p. 388) 1865; Biography, Brit. J. Surg. **6**: 157-159, 1918-1919.

2. Thomson, A.: Observations on the Circumscribed Abscess of Bone (Brodie's Abscess), Edinburgh M. J. **19**:297-309, 1906.

3. Gross, H.: Zur Kenntnis des osteomyelitischen Knochenabscesses der langen Röhrenknochen, in besonderer Berücksichtigung seines anatomischen Verhaltens, Beitr. z. klin. Chir. **30**:231-298, 1901.

4. Brickner, W. M.: The Treatment of Chronic Bone Abscesses by Simple Evacuation Through a Small Drill Hole. Its Application in Non-Sterile Abscesses, Jour. Bone and Joint Surg. **5**:492-500, 1923; Chronic Medullary Abscess of the Long Bones, Ann. Surg. **65**:483-490, 1917.

been able to collect twenty-six additional cases from the literature,[5] and have observed thirteen in the Mayo Clinic. Reference has been made by various authors to cases which they have not reported in detail. The sparsity of reported cases is probably owing to failure to differentiate the condition from chronic osteomyelitis, bone cysts, and other similar conditions.

## ETIOLCGY

It has been shown fairly conclusively that the staphylococcus is usually the infecting organism. Bacillus typhosus on rare occasions has been found in the abscess.[6] In two modern textbooks on surgery,[7] the condition has been erroneously referred to as "sometimes of tuberculous origin." In our thirteen cases the staphylococcus was found in three cases; examination for organisms was negative in five cases, and no

TABLE 1.—*Location of Abscess in 200 Cases*

| Location | Thomson's Cases | Other Collected Cases | Mayo Clinic Cases | Total |
|---|---|---|---|---|
| Upper end of tibia | 63 | 6 | 4 | 73 |
| Middle of tibia | 2 | 1 | 0* | 3 |
| Lower end of tibia | 42 | 9 | 8 | 59 |
| Tibia (portion not stated) | 1 | 3 | 0 | 4 |
| | | | | —139 |
| Lower femur | 18 | 2 | 1 | 21 |
| Humerus | 18 | 2 | 0 | 20 |
| Radius | 4 | 1 | 0 | 5 |
| Ulna | 2 | 0 | 0 | 2 |
| Not stated | 11 | 2 | 0 | 13 |
| | | | | — 61 |
| Total | .. | .. | .. | 200 |

* Not considered as Brodie's abscess.

such examination was recorded in five cases. In five cases, a special search was made by microscopic examination and animal inoculation for evidence of tuberculosis, with negative results. Evidence of pulmonary tuberculosis was found in only one case, in which inactive tuberculosis of both upper lobes was demonstrated by the roentgen ray. Clinically,

5. Bancroft, F. W.: Two Cases of Localized Bone Abscess (Brodie's Abscess), One Case of Osteitis Fibrosa Cystica, and One Case of Chronic Traumatic Osteomyelitis, Surg. Clin. N. America **1**:1773-1803, 1921. Dowd, C. N.: Staphylococcus Bone Infection, Ann. Surg. **44**:111-112, 1906. Martin, W.: Chronic Suppurative Osteomyelitis, Ann. Surg. **66**:254, 1917. Miller, A. M.: Note on Isolated Abscesses of Long Bones, Ann. Surg. **62**:460, 1918. Symonds, C.: Chronic Abscess of Bone and Its Treatment, Guy's Hosp. Gaz., 1915.

6. McWilliams, C. A.: Central Bone Abscess, Ann. Surg. **74**:568-578 (Nov.) 1921.

7. DaCosta, J. C.: Modern Surgery, General and Operative, Ed. 8, Philadelphia, W. B. Saunders, 1919, p. 569. Skillern, P. G., Jr.: Interesting Surgical Cases (Case 2) Internat. Clin. Series 24 **1**:174, 1914.

the chest findings were negative, and guinea pigs inoculated with the material from the abscess gave negative results.

In 145 of Thomson's 161 collected cases, there was a history of previous osteomyelitis in 122. Such a history was obtained in only two of our thirteen cases. The location of the abscess was indicated in all but thirteen of the 161 cases collected by Thomson, in the twenty-six miscellaneous cases collected by us, and in the thirteen cases in our own series (Table 1).

Just why the tibia is affected so much more often than any other bone is not known. If, as Ogilvie [8] suggests, "the primary lesion is a juxta-epiphyseal strain, a partial injury at the very vascular area plus general lowered resistance in the presence of foci elsewhere," we could explain this localization as being due to greater frequency of trauma to the tibia. The further observations that twelve of our thirteen cases occurred in men averaging twenty-one and one-half years, and that in six, a history of injury was given, would all serve to direct attention to trauma as a factor in the cause of the lesion. It must be conceded, however, that trauma plays only a minor or contributory part.

### SYMPTOMS AND ROENTGEN-RAY FINDINGS

Brodie's [9] description of the lesion is as follows:

> When the tibia is enlarged, from a deposit of bone externally, when there is excessive pain, such as may be supposed to depend on extreme tension, aggravated at intervals, and these symptoms continue and become aggravated and do not yield to treatment, you may reasonably suspect the existence of abscess in the center of the bone. You are not to suppose that there is no abscess because the pain is not constant. On the contrary, it very often comes on only at intervals, and in one of the cases which I have related there was an intermission of seven to eight months. After it has lasted a number of years, the pain never entirely subsides, still it varies.

Either extremity of the tibia may be affected, and occasionally the ends of other long bones. Pain and enlargement of the bone are the most prominent symptoms. The pain is often worse at night, and is sometimes referred to the adjacent joint. On close questioning, the patient can usually be made to recall mild and transient pains occurring years before, which were passed over at the time as "rheumatic" or as "growing pains." In exceptionally severe cases, the patient may become emaciated and greatly weakened as a result of suffering and loss of sleep. As a rule, there is little or no evidence of a general reaction; the temperature is normal, or elevated not more than one degree, and there is seldom any leucocytosis.

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8. Ogilvie, W. H.: Central Abscess of Bone, Clin. J. **51**:385-389, 1922.

9. Brodie, B.: Brodie's Tumor and Brodie's Abscess, Eponyms, Brit. J. Surg. **9**:334-337 (Jan. 22) 1922.

Thomson [10] records a remarkable case of Brodie's abscess, the specimen of which is in the museum of the Royal College of Surgeons of Edinburgh. In the upper end of this tibia is a cavity with a capacity of more than 500 c.c. communicating externally by an opening 2.5 cm. in diameter. It is said that this patient had been able to use his limb quite well, and that he would periodically remove a plug of wood that he kept in the opening, and empty out the contents of the cavity.

The abscess may occasionally rupture externally, or into the joint. But this is a very unusual complication, as is the formation of a sequestrum, or invasion of the shaft.

TABLE 2.—*Summary of Thirteen Cases of Brodie's Abscess Observed at the Mayo Clinic from Jan. 1, 1914, to May 1, 1924*

| Case | Age | Sex | Leukocytes | Duration, Years | Bone Involved | Past Illness or Injury | Bacteria |
|---|---|---|---|---|---|---|---|
| 1 | 21 | M | 9,400 | 4 | Upper third tibia | Pneumonia eight years before examination; influenza one year before | Not recorded |
| 2 | 21 | M | 9,000 | 12 | Upper third tibia | Influenza; fracture twelve years before | Negative |
| 3 | 14 | M | 9,000 | 3 | Upper third tibia | Nephritis; cleared up..... | Staphylococcus albus |
| 4 | 11 | M | 7,000 | 0.67 | Lower third tibia | Osteomyelitis five years before; influenza just before onset | Not recorded |
| 5 | 14 | M | 11,100 | 5 | Lower third tibia | Influenza four years before | Staphylococcus albus |
| 6 | 37 | M | 6,400 | 10 | Lower femur | Typhoid at fifteen..... | Negative |
| 7 | 21 | F | 4,800 | 8 | Lower tibia | Measles and smallpox three years before | Negative |
| 8 | 13 | M | 8,600 | 3 | Lower tibia | Influenza one year before with aggravation of symptoms seven months before | Negative |
| 9 | 29 | M | 10,800 | 14 | Lower tibia | Influenza; osteomyelitis seventeen years before | Not recorded |
| 10 | 27 | M | 9,900 | 12 | Lower tibia | Smallpox; sprain twelve and eight years before | Negative |
| 11 | 15 | M | 12,200 | 5 wks. | Lower tibia | Sprain five years before; kick five weeks before | Not recorded |
| 12 | 17 | M | 7,800 | 2 | Lower tibia | Influenza one year before; sprain two years before | Staphylococcus albus |
| 13 | 38 | M | 8,800 | 4.5 | Lower tibia | Kicked four and one-half years before; scarlet fever two weeks after kick | Not recorded |

In our cases there was a history of symptoms being present for from five weeks to fourteen years. In a reported case there was a history of sixty years' duration.[11]

The roentgen ray generally reveals an increase in the diameter of the bone, gradually fading off to the normal, periosteal thickening, often sclerosis of the cancellous bone, and usually a sharply outlined cavity,

10. Cathcart, C. M.: Catalog of Royal College of Surgery of Edinburgh **1:** 243 (quoted by Thomson, p. 302).

11. Choyce, C. C.: A System of Surgery, Ed. 2, London, P. H. Hoeber, **3:** 743-745, 1923.

showing up as a rarefaction of from 2 to 4 cm. in diameter. If such sclerosis is very extensive, it may obscure the area of rarefaction, and make the diagnosis more difficult.[12]

### PATHOLOGY

A thickened periosteum, beneath which the bone is hard and sclerosed, is usually revealed by exploration. The degree of hardening of bone varies somewhat, ranging from nearly normal to a degree of hardness approaching eburnation. On chiseling through this bone, a cavity is encountered which, in the earlier stages, is small, smooth-walled, and lined with a vascular membrane, resembling granulation tissue. Within this cavity is generally found a liquid material which may vary from a thin, straw colored fluid to thick creamy pus. Occasionally, there is no definite cavity, but only an area of soft granulation tissue, the precursor of a cavity. The abscess, or area of softening, is usually near the epiphysis, and there is a greater tendency for it to gravitate toward the diaphysis. A tuberculous abscess, on the other hand, is even closer to the epiphyseal line, and tends rather to extend outward or toward the joint.

For descriptions of the lesion, other than that which can be seen at operation, we must turn to the earlier writers who occasionally amputated, or to those who have had the good fortune to fall heir to a specimen from a patient who had died from some other cause. Modern surgical methods preclude the obtaining of a complete specimen.

Thompson describes three stages: (1) the quiescent focus, (2) the mature abscess, (3) rupturing and discharge externally. The quiescent focus is represented by those cases in which a definite cavity is not found, but only a jelly-like mass, devoid of tissue elements and surrounded by a lining membrane. These findings Thomson explains on the assumption that when the staphylococci gained admission to the bone-marrow, they were walled in by granulation tissue, which later organized into fibrous tissue.

The mature stage is the one most commonly encountered at operation. It is in these cases that there is characteristic intermittent pain, which may be accounted for by variations in the pressure within the cavity. That this would be capable of causing such pain is borne out by the observation of an early writer that the patient complained of very severe pain when the lining membrane was touched during operation. In the third stage, the tension is relieved by the free discharge

12. Baetjer, F. H., and Waters, C. A.: Injuries and Diseases of the Bones and Joints; Their Differential Diagnosis by Means of the Roentgen Rays, New York, P. B. Hoeber, 1921, p. 168. Bryant and Buck: Textbook of Surgery, New York, Wm. Wood, **3**:289-316 and 321, 1906-1911 (quoted by McWilliams, p. 578).

of pus, with a consequent cessation of pain and termination of enlargement of the cavity.

Situated as the abscess is, in the spongy cancellous type of bony tissue, there is little tendency to the formation of a sequestrum such as occurs when destructive infectious processes involve the harder and more resistant types of bone. Occasionally, a small sequestrum is found.

Fig. 1 (Case 2).—Brodie's abscess of the upper end of the tibia, of twelve years' duration: left, from front; right, from side.

## DIFFERENTIAL DIAGNOSIS

The differential diagnosis is not difficult. It is important that the condition be kept in mind when a patient complains of pain in or around the knee, ankle, and possibly the elbow or shoulder joint, particularly if there is enlargement of the adjacent end of one of the long

Fig. 2 (Case 4).—Large abscess of the lower end of the tibia, of eight months' duration. Note beginning sclerosis at margin of abscess, and beginning of thickening of bone.

Fig. 3 (Case 4).—Lateral view of Figure 2.

bones. The distinction from medullary abscess is made on the location of the latter, at or near the center of the long bone, rather than at either end.

Chronic sclerosing osteitis [13] involves the medullary portion of the bone rather than the ends; it is often accompanied by fever and leuko-

Fig. 4 (Case 5).—Abscess of the lower end of the tibia with considerable sclerosis of bone obscuring the rarefied area.

cytosis, and gives a rather typical roentgen-ray picture of spindle-shaped enlargement of the shaft, thickening of the cortex, and narrow-

13. Henderson, M. S.: Chronic Sclerosing Osteitis, J. A. M. A. **82**:945-949 (March 22) 1924.

ing of the medullary cavity. The typical area of rarefaction is absent in the roentgenogram.

Sarcoma offers difficulties of diagnosis, especially if the case is of short duration. It often involves the end of the long bone, but the enlargement is more rapid, and the pain less severe; it occurs more

Fig. 5 (Case 5).—Lateral view of Figure 4, showing more clearly the abscess cavity.

often in the child than does Brodie's abscess. Enlarged veins on the surface, a not uncommon finding in sarcoma, were not noted in any of our cases. Roentgen-ray examination fails to reveal the localized area of rarefaction, but shows a process more extensive and more destructive in character; there is usually a tumor with an irregular, blurred outline.

Cases of syphilitic periostitis or osteitis practically always show characteristic serum reactions, and such a diagnosis would be supported by a history of a primary lesion and the finding of other evidence of syphilis.

Bone [14] cysts lack the pain and tenderness, and, rather than an increase in thickness and density of the surrounding bone, there is likely to be a thinning. Confusion of this condition with rheumatism is usually due to lack of careful examination.

Tuberculous abscess is difficult to diagnose, and has often been confused with Brodie's abscess. In some cases, it can only be ruled out by negative findings, on microscopic examination and animal inoculation. The tuberculous abscess is smaller and more strictly localized, with, as a rule, very little thickening of periosteum or bone, and either it has a tendency to early perforation of the epiphyseal plate with involvement of the joint, or it may work laterally and rupture. The wall of the bone surrounding the tuberculous abscess is rarely as sclerotic as it is in the infectious type.

### TREATMENT

Brodie treated his first patient by amputation. His observation of the lesion showed him the needlessness of such a radical procedure, and his second patient was successfully treated by trephining. Since then partial resection, packing, curettage with attempted chemical sterilization [15] followed by primary or secondary closure, or mere drainage of the cavity with immediate closure,[16] have all been advocated and used.

The treatment will depend somewhat on the location, size and activity of the abscess. In cases in which there is a small and more or less inactive cavity, aseptic drainage and primary closure will often result in cure. If the cavity is large, it may be desirable to remove more of the surrounding bone and attempt to obliterate the cavity by dropping in the overlying soft tissue. If, on the other hand, there are signs of activity, such as considerable fever, leukocytosis, or rapid aggravation of symptoms, drainage would be indicated. Surgical solution of chlorinated soda may be used to keep the cavity clean after drainage, promote healthy granulations, and thus gradually obliterate the cavity.

### MAYO CLINIC SERIES

Twelve of the thirteen patients were males. The oldest patient was aged 38, the youngest 11, and the average age was twenty-one and one-

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14. Ashhurst, A. P. C.; Bromer, R. S., and White, C. Y.: Cystic Disease of the Bones, Arch. Surg. **6**:661-730, 1923. Meyerding, H. W.: Cystic and Fibrocystic Disease of the Long Bones, Am. J. Orthop. Surg. **16**:253-276, 367-382 (No. 9) 1918.

15. Bevan, A. D.: Brodie's Abscess, Surg. Clin., Chicago **3**:743-748, 1919.

16. Brickner, W. M.: Chronic Bone Abscess; Its Treatment by Simple Evacuation Through a Drill Hole, Surg., Gynec. & Obst. **35**:84-88, 1922.

half years. The duration of the symptoms ranged from five weeks to fourteen years, with an average of seven years. All but two of the patients came to the Clinic because of pain; they complained chiefly of enlargement of the bone, with pain as a minor factor. One patient with involvement of the lower end of the femur gave a history of partial locking of the knee, occurring six or seven times during the preceding year. Although seven of the patients complained of pain in the joint rather than in the end of the bone, only three of these had limitation of motion, in one of whom it was due to arthritis.

In practically all the cases, with the onset was associated intermittent pain, mild at first, worse at night, gradually tending to become more severe and more nearly constant. In all, there was enlargement of the end of the bone. In six cases, there was associated tenderness with moderate increase in local temperature and slight redness. In five cases, the temperature ranged between 99 F. and 100 F., and in one case, it was 100.2 F. The highest leukocyte count observed was 12,200; in six cases, it was between 8,000 and 12,000, and in the remainder, below 8,000.

In eight of the thirteen cases, the results following operation were entirely satisfactory. In four cases, drainage developed one, two, five and seven months respectively after operation, and in one case in which no abscess had been found at operation, there was recurrence of symptoms after a period of two months. The treatment was simple drainage with primary closure in two of the cases, which were quiescent. In those with more signs of activity, curettage and drainage were employed, followed in some cases by the use of phenol and alcohol or iodin to swab out the cavity, by iodoform pack, or by the use of dichloramin-T (Table 2).

## COMMENT

We have accepted as Brodie's abscess only cases in which involvement of the cancellous tissue at the ends of the long bones was evident, as we believe it is only in these cases that the true picture as presented by Brodie is found.

Speculation as to the possible cause for the activation of the process has led to various theories. Everyone is cognizant of the ability of bacteria to lie dormant in a focus for years, as is illustrated by the finding of typhoid bacilli in the gallbladder years after the acute infection, and by cases of chronic osteomyelitis in which there are periods of quiescence extending over many years.[17] Given, then, a dormant focus in a bone, in which the general resistance is just great enough to hold the organisms in check, it is only necessary for the local or general

17. Trotter, W.: Diagnosis of the Common Swellings of the Long Bones, Clin. Jour. **29**:198-208, 1907.

resistance to be lowered in order that the process may become active. Six of our thirteen patients date their symptoms from local trauma; in three, the onset very definitely followed an attack of influenza, and in one it followed an attack of scarlet fever after local trauma.

## CONCLUSIONS

1. The symptoms of Brodie's abscess are characteristic, and may be explained on a pathologic basis.

2. It is a focus of infection situated in the spongy portion of the long bones near the epiphyseal line, arising either as a remnant of a former osteomyelitis, or of unexplained origin.

3. There is often a period of latency which may be quite long, during which the resistance of the body and the local wall of sclerosed bone at the abscess margin holds the process in abeyance.

4. It may burst into activity as a result of lowered resistance, due in some cases to trauma or acute infection.

5. The occasional complications are: (*a*) sinus formation with sequestrum, (*b*) extension into the diaphysis, (*c*) effect on general health, and (*d*) marked enlargement.

6. The condition is not tuberculous in origin.

7. Surgical treatment suited to the location in the bone, and to the type of the abscess, results in cure in the majority of cases. Recurrence may occasionally be met with after surgical treatment.

# THE USE OF FREE TRANSPLANTS OF FASCIA AS LIVING SUTURES IN THE TREATMENT OF HERNIA*

W. E. GALLIE, M.D.

AND

A. B. LEMESURIER, M.D.

TORONTO, CANADA

Recurrence of hernia after presumably satisfactory operations is much more frequent than is ordinarily supposed. The records of our army hospitals have demonstrated a very surprising percentage of failures among patients who were otherwise fit for general service and who, owing to their youth and muscular development, are considered the most satisfactory risks for operative treatment. This applied not only to direct inguinal hernias and to ventral hernias resulting from wounds or previous abdominal operations, but also to simple oblique inguinal hernias. The opportunity afforded by the collection of a large number of these cases over a short period of time led us to a clinical and experimental investigation of the subject, which has resulted in the formulation of some conclusions as to the cause of the failures as well as in the elaboration of a remedy which we think will lead to their reduction.

After operations for the cure of oblique inguinal hernia, recurrence takes place in a variety of ways. In some cases, the hernia reappears in the cord and is probably due to incomplete removal of the sac. In many, however, the recurrence is in the form of a direct hernia, and must be accounted for in most instances by damage done to the abdominal wall during the first operation. In looking for an explanation of the development of these direct hernias, we found that in all our cases the external ring was wide open, a condition that may have resulted from improper suture of the split external oblique aponeurosis, or from stretching of the scar in the line of union after the sutures had been absorbed. Whatever the cause, the result was the same, namely, the production of an area in the anterior wall of the inguinal canal that was unsupported by aponeurosis, through which the fascia transversalis and peritoneum were forced during periods of increased abdominal pressure. In all the operations, the internal oblique muscle and conjoined tendon had been sutured to Poupart's ligament either in front of or behind the cord. In some instances, at the time of the second

*Read before the American Surgical Society, Rochester, Minn., May 31, June 1 and 2, 1923.

operation, there was no evidence to show that such an operation had ever been performed, and it was evident that the muscle and tendon must have returned to their normal positions soon after the sutures had been absorbed. In the majority, however, the muscle had adhered in part, particularly toward the outer end of the canal. Toward the inner end either the muscle had completely separated from Poupart's ligament, leaving it smooth and shiny as it is in its natural condition, or the hernia had pushed through the muscle itself, close to the ligament, leaving a few bundles of muscle firmly adherent to it. The most important observation, however, was that in no case did we find any evidence of adhesion between the conjoined tendon or the sheath of the rectus and the ligament. In every instance, the aponeurotic structures that had been sutured to the ligament had returned to their normal positions, leaving an area of weakness which the original operation had been designed to strengthen. It was through this portion of the inguinal canal that the hernias protruded. From these observations, we were convinced that the simple side-to-side suture of the muscle and the conjoined tendon to Poupart's ligament was an untrustworthy method of supporting the posterior wall of the canal, and it has raised in our minds a doubt as to whether, even in successful operations, it plays an important part in the cure.

Our observations on direct hernias that had recurred were similar to those made on oblique hernias. In addition to the suture of the internal oblique muscle and conjoined tendon to Poupart's ligament, the records showed that, in many instances, the sheath of the rectus and the abdominal aponeurosis had also been dragged down to the ligament and fastened there with heavy chromicized catgut or kangaroo tendons. In none of the cases had permanent adhesion taken place, and the recurrence had developed for precisely the same reason that the original hernia had developed. Our experience with direct hernia was such as to convince us that all of the methods employed to close the posterior wall of the canal were exceedingly uncertain, and that even successful operations were largely a matter of chance.

The ventral hernias recurring after operation simply showed that the muscular or aponeurotic structures that had been sutured had failed to heal together firmly, and our operations showed a protrusion of parietal peritoneum covered with more or less thinned-out scar tissue. In some of the cases the aponeuroses had been simply drawn into edge-to-edge apposition with heavy absorbable sutures, but in others the various layers had been carefully overlapped. Our conclusion was that here, likewise, the ordinary operations leave altogether too much to chance and that some radical change must be made in the operative technic before a cure can be expected with confidence.

Among the ventral hernias admitted for treatment were several in which large areas of the abdominal wall had been destroyed by shell wounds, and in which huge hernias were covered only by a thin layer of scar. These cases were considered inoperable and the patients had been sent to hospitals to be supplied with some form of abdominal belt. It was these cases in particular that set us thinking of ways by which large gaps in the abdominal wall might be closed, and that led to the experimental investigation of which this paper contains a review.

The experiments were performed on dogs and rabbits. They consisted of a series of operations, in some of which simple wounds in fascia, aponeurosis and muscle were sutured with catgut and linen, and in others pedunculated and free transplants of aponeurosis and fascia were sutured into gaps in these structures. A detailed account of these experiments has been given elsewhere,[1] but the findings may be summarized as follows:

### EXPERIMENTAL FINDINGS

1. Wounds in fascia and aponeurosis, when sutured in edge-to-edge apposition with absorbable sutures, heal together by means of delicate scar tissue, which develops from the areolar membrane investing the surfaces and from the loose connective tissue stroma separating the bundles of fibers. This scar is not strong, and, if the wound is subjected to strain, it slowly stretches, leaving a gap between the edges of the wound, which is filled with areolar membrane continuous with that on the surfaces of the aponeurosis or fascia (Figs. 1 and 2).

2. When linen sutures are used, the tendency toward opening up of the wound when it is under strain is not so marked. The sutures themselves assist in holding the edges together, and the scar in the region of the sutures is more abundant and denser than when catgut is used. In many cases, however, the linen cuts out and the edges of the wound spread apart exactly as they do when absorbable sutures are employed.

3. The simple overlapping of the edges of the wound adds little or nothing to the solidity of the healing. By this procedure the overlapped edges are not placed in actual contact, as the areolar tissues on the surfaces are interposed. These adhere lightly to one another, but any degree of strain causes the edges of the wound to draw apart. The process of overlapping increases the degree of strain, thus tending to defeat its own object.

4. If, before overlapping the edges of the wound, the areolar membranes on the surfaces are thoroughly removed and the areas placed in contact scraped and otherwise damaged, the degree of adhesion is much increased. The scar between the overlapped layers is more dense, and it has a deeper grip on them than it would have under any other circumstances. Prolonged and unusual strain, however, results in stretching of the scar just as in the other methods of suture.

5. Side-to-side suture of undamaged folds of aponeurosis, such as is done in the suture of the conjoined tendon to Poupart's ligament, never results in permanent adhesion even under the slightest degrees of strain.

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1. Gallie, W. E., and LeMesurier, A. B.: Living Sutures in the Treatment of Hernia, Canad. M. A. J. **13**:469-480 (July) 1923.

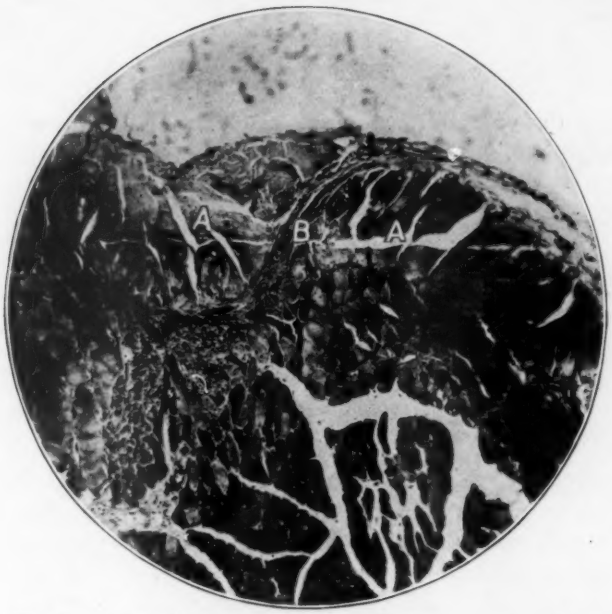


Fig. 1.—Healing of incision in aponeurosis, three weeks after suture: *A*, aponeurosis on either side of incision; *B*, new tissue uniting the edges of the cut. This new tissue resembles and seems to be derived from the areolar covering of the aponeurosis rather than from the aponeurosis itself. The edges of the incision are in apposition because they are not under tension.

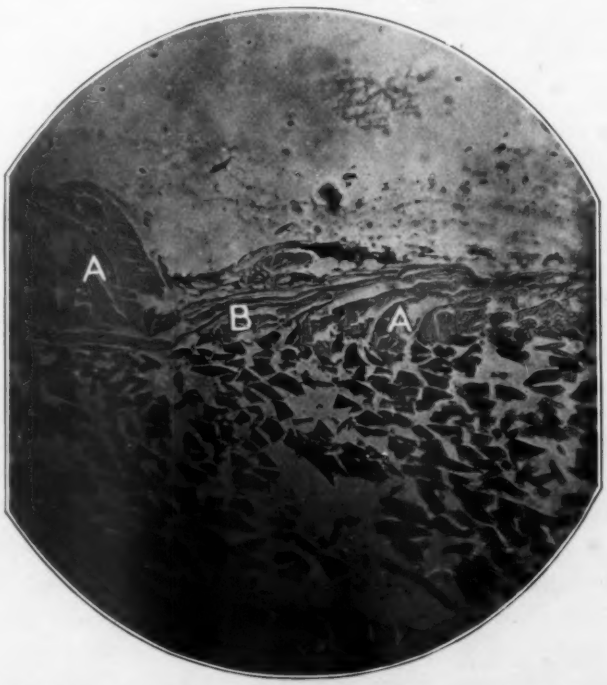


Fig. 2.—Healing of incision in aponeurosis thirteen months after suture. *A*, aponeurosis on either side of incision; *B*, new tissue uniting the edges of the cut. The wound was sutured under slight tension, and the new tissue forming the line of union has stretched, allowing the edges of the wound to separate.

6. Scarification of the surfaces of the folds before suture increases the degree of adhesion, but not to any great extent.

7. Side-to-side suture of undamaged muscles which, in contracting, tend to draw apart, such as suture of the two recti muscles in the middle line of the abdomen, results in the lightest possible adhesion, which disappears in a very short time.

8. Preliminary scarification and scraping of the sides of the muscle before suture adds greatly to the solidity of union. This is due to the injury done to the connective tissue stroma of the muscle, and is responded to by the formation of a firm scar, which has a solid grip on the depths of the muscle.

9. Side-to-side suture of muscle and aponeurosis, as in the suture of the internal oblique muscle to Poupart's ligament, results in union of practically no strength. The strength of union is increased by preliminary damage to the sutured surfaces, but to a much less degree than in the suture of muscle to muscle. This is due to the fact that the smooth, shiny surface of the aponeurosis allows very little opportunity for a scar to get a solid grip of it.

10. Pedunculated or free transplants of fascia and aponeurosis, if placed in such a position that they can receive an adequate supply of lymph, continue to live unchanged.

11. Such transplants, when they are used to fill anatomic defects, such as occur in inguinal and ventral hernias, heal to the structures to which they are sutured in exactly the same manner that has already been described in the healing of simple wounds in these tissues. If the edges are accurately brought together or are overlapped without scarification, the degree of adhesion is only that which is provided by a delicate areolar tissue. If they are overlapped and subjected to considerable damage, they unite by a generous scar. In the former case, the slightest strain will cause the separation of the edges, but in the latter the union is more solid and often tolerates what may be considered normal degrees of strain. If the strain, however, is unusual or prolonged, the scar behaves exactly as it does elsewhere under similar conditions, and slowly stretches (Fig. 2).

## CONCLUSIONS

From our clinical experience, therefore, and from the information derived from our experiments we have come to the conclusion that operations designed to produce permanent adhesion between aponeurotic structures having natural tendency to separate are defective in principle, if they depend solely on the process of healing in the line of suture. Herein lies the explanation of the high percentage of recurrences in large ventral hernias and in direct inguinal hernias, even when the operations have been performed by most competent hands. Before this percentage of failures can be eliminated, some method must be discovered of removing the element of chance, which always attends dependence on the strength and resistance of scar.

It was while we were considering means of overcoming this difficulty that the idea presented itself to us of using transplants of fascia lata as living sutures, which might permanently hold the edges of hernial rings together without depending upon the natural process of repair. Before trying this method on patients, we performed a series of experi-

ments on animals, in which strips of fascia, a quarter of an inch wide, were removed from the back and then used as sutures to hold together the edges of the opening so produced. These edges were left about a quarter of an inch apart in order that the whole test might be placed on the sutures without any assistance from scar tissue. The specimens were recovered at intervals of from one week up to two years. As in the case of ordinary patch transplants, the fascial sutures continued to live practically unchanged. By means of a special apparatus we were able to demonstrate that their tensile strength remains constant, and by fastening to them black silk markers at accurately measured intervals,

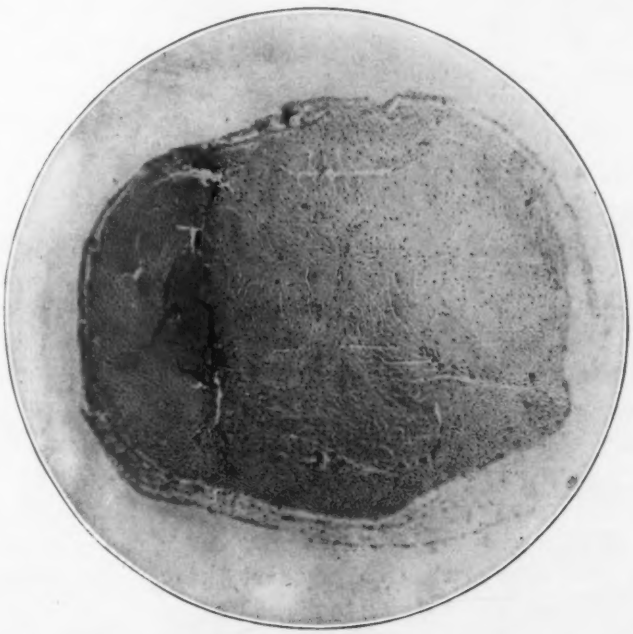

Fig. 3.—Cross-section of fascial suture after six weeks. The suture at this stage resembles tendon in that it consists of a mass of parallel fibers separated into bundles by delicate septa of vascular connective tissue.

that they neither stretch nor contract. As a result of passing through a needle hole, they become folded into rounded cords, which soon become surrounded with a delicate vascular membrane, such as is normally on the surface of fascia. From this membrane, processes of similar tissue project inward among the folds of the suture, and when the early inflammatory reaction has subsided and the newly formed connective tissue has contracted down to its normal adult condition, the suture appears in cross-section like ordinary normal tendon (Fig. 3). In all the experiments the suture was successful in holding the edges of the

gap in the fascia at the same distance from one another as at the time of the operation, in spite of the fact that the animals were under no restraint. It was only necessary to make certain that the suture obtained a solid grip of the edges of the opening to assure the success of the operation.

From these experimental findings, we felt sure that in fascia lata we would find a suture material which would be ideal for the closure of hernia. It is easy to obtain in long strips and in any quantity, and its removal results in no disability. It is extraordinarily strong, being simply flattened tendon, and its smooth, glistening surfaces make it admirable for use as sutures. We have accordingly employed it whenever we have had reason to doubt the certainty of the ordinary methods in establishing a cure and as time goes on it is being employed with steadily increasing satisfaction.

### OPERATIVE TECHNIC

From our experimental and clinical experience, certain points in technic have been evolved that have proved themselves of assistance. Great saving of time can be obtained by having two operators, one to attend to the hernia and the other to look after the sutures. The sutures are obtained through a long incision on the lateral aspect of the thigh, slightly posterior to the horizontal midplane. Here, the fascia is most tendinous in character. After the surface of the fascia is thoroughly cleared of fat and areolar tissue, a longitudinal slit is made in it with a knife, and, with a pair of blunt scissors, the fascia is ripped to the required length, much as a draper rips a bolt of cotton. A second incision is then made, a quarter of an inch lateral to the first, and one end of the suture thus prepared is cut free. This end is now threaded into a large-eyed needle and tied securely into it with catgut or silk. This is necessary, owing to the slippery character of the fascia, which causes it to slide readily out of the needle. As many sutures as are required are prepared in this way, and the wound is then drawn together with towel clips until they are called for. By this means, the handling of the sutures is reduced to a minimum and they are not allowed to become dried by exposure to the air. When the first suture is required, the needle is picked up with forceps, and the strip of fascia quickly lifted out of its bed and detached. A catgut ligature is tied around its terminal end to prevent it from fraying and it is transferred to the hernial wound. The needle is then passed through whatever solid aponeurotic structure is available, then through the terminal end of the suture close to the catgut ligature and drawn taut. In this way a sort of slip-knot is produced, which makes an excellent anchor for the stitch (Fig. 4, *A*). A running suture is now made, which draws

together the strongest aponeurotic tissue in the neighborhood. It is wise to introduce some form of lock-stitch about every third or fourth bite of the needle, owing to the slippery nature of the fascia; we have found the most useful one to be a single knot combined with transfixion (Fig. 4, *C*). When the first suture has been used up, a second one is started by fastening it to the first, in the manner used in stringing a tennis racquet (Figs. 4, 5 and 6), and the sewing is proceeded with as before. To terminate the suture, we insert a final lock-stitch and then, detaching the needle, split the strip of fascia into two strands, one of which is passed around the main portion of the suture and tied to the other in a triple knot. This knot is securely fastened by a

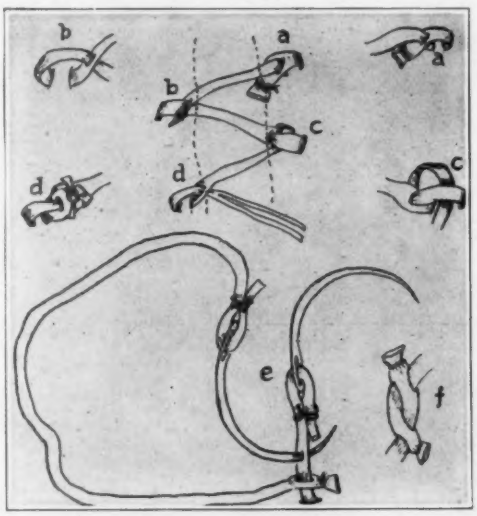


Fig. 4.—Diagram showing method of using fascial suture: *a*, method of anchoring suture at its commencement; *b*, method of fixing each stitch by simple transfixion; *c*, method of fixing stitch by transfixion combined with a single hitch; *d*, method of ending the suture by passing it through itself, splitting and tying the ends in a triple knot and transfixing the whole by a catgut or silk suture; *e*, method of joining a new suture to one that has been expended, and *f*, join completed.

catgut ligature which transfixes it, with a view to preventing it from becoming untied (Fig. 4, *D*).

The foregoing description of technic applies particularly to those operations in which it is possible to draw the edges of the hernial ring together without too great tension. In these cases, when the process of healing is complete, the edges of the ring are held together by both sutures and scar tissue, so that the number of sutures need rarely be more than two. When the edges cannot be drawn together, however,

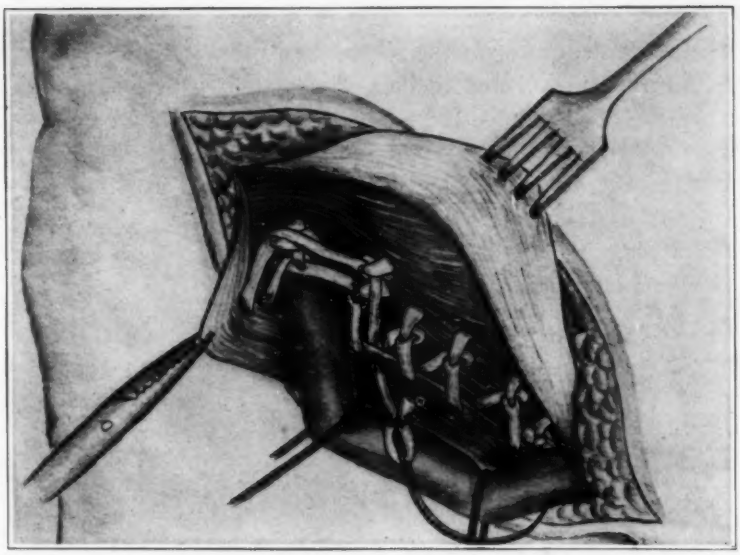

Fig. 5.—Repair of inguinal hernia; insertion of first row of fascial sutures, fixing the internal oblique to Poupart's ligament and reinforcing the internal ring.

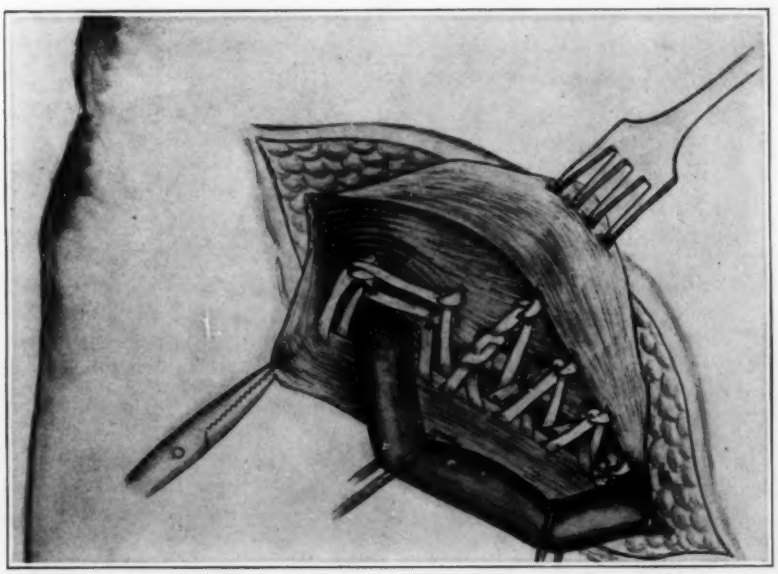

Fig. 6.—Repair of inguinal hernia; insertion of second row of fascial sutures. These are fixed above by a deep bite into the line of junction of the internal and external oblique aponeuroses, and below into Poupart's ligament; they do not drag these structures into contact, but fill in the interval between them.

the whole strain comes on the sutures and it is necessary, therefore, to make absolutely certain that they get a solid grip of the neighboring aponeuroses and muscles and to use a sufficient number of sutures to fill up the gap completely, thus preventing a hernia from forcing its way through one of the chinks. In such circumstances, we weave the sutures across the opening as in the darning of a sock.

In operations on the inguinal canal, the same general technic is observed in all types of hernias. After the sac has been disposed of, special care is taken to expose and clear the conjoined tendon and rectus sheath at their insertion into the pubic bone and also the point of attachment of Poupart's ligament. The split external oblique is turned back until the glistening abdominal aponeurosis is in full view. The fascial suture is securely anchored into the conjoined tendon and sheath of the rectus close to their insertion into the bone, and then passed through the periosteum of the spine of the pubis and the inner end of Poupart's ligament. When the suture is drawn taut, the weakest spot in the posterior wall of the canal is permanently closed, so that no hernia can force itself through. The suture is then continued in an outward direction, closing the internal oblique muscle down to the recurved edge of Poupart's ligament, behind the cord, until the position of the internal ring is reached. Here a lock-stitch is inserted and the suture is continued to the outer side of the cord and a stitch or two taken to lend additional support. In this way the cord is surrounded in the region of the internal ring by a strong ring of fascia, which should permanently prevent recurrence into the cord (Fig. 5). So far, the operation very closely resembles the various well-known operations, with the exception that fascia is substituted for catgut or other suture materials. We are of the opinion, however, that for direct hernias this is not sufficient, as a hernia may recur through the muscle itself. To prevent such a mishap, we insert a second layer of sutures, which weave the abdominal aponeurosis and the rectus sheath to Poupart's ligament. This suture is continuous with the first, starting at the internal ring and ending at the spine of the pubis (Fig. 6). No attempt is made to drag the abdominal aponeurosis down to Poupart's ligament, as in most cases this would result in tearing the ligament to pieces, but the sutures are simply drawn sufficiently taut to make them lie flat. They are interwoven with the first row of sutures so that a solid mat results, such as is produced in weaving a basket. It is on this second row of sutures that we depend for strength in closure of the defect, as the anchorage in the aponeurosis and the ligament is absolutely dependable and we have no fears in regard to the suture material itself. The only real virtue in the deep sutures is that they permanently cover the peritoneum with a layer of muscle which will prevent the possibility of a hernial protrusion through the chinks in

the superficial layer. When the suture has been securely tied, we feel that the important part of the operation has been completed. What one does to the split external oblique appears to be of relatively little importance, as it will never be subjected to any severe strain now that the defect in the abdominal wall is closed off. Sometimes we have simply sewn it over the cord with catgut, but in patients who have already had two or three operations we have used another narrow fascial suture. In such cases, the external oblique aponeurosis in the region of the external ring is usually very defective, and we have taken pains to make a new aponeurosis in this area by running the suture from the anterior aspect of the rectus sheath to Poupart's ligament and to construct a new external ring, which lies in front of the pubic bone, so that it is not subject to strain during increases of intra-abdominal pressure.

With this type of operation, we have had an experience extending over four years. During that time, about sixty operations have been performed on patients, the majority of whom have been operated on once, twice or three times previously. In most instances, the hernias were of the direct variety when referred to us, although many had originally been oblique. In this series, after the living suture operation, there have been no recurrences. We have sufficient confidence in the method, therefore, to recommend it to this society.

It has been brought to our attention that the principle of using living tissues as suture material has been suggested before. Years ago, Vulliet described a method of nephropexy in which he sutured the capsule of the kidney to the abdominal wall by means of a pedunculated strand of the tendon of the longissimus dorsi. In 1901, Dr. L. L. McArthur described a method of procedure in inguinal hernia, in which pedunculated strands of the aponeurosis of the external oblique were used in place of catgut in suturing the internal oblique muscle to Poupart's ligament and in closing the split external oblique itself. As far as we know, however, no one has suggested the use of free transplants of fascia lata as suture material, and no one has used the principle of filling defects in the abdominal wall with a living aponeurotic filigree.

While the results in inguinal hernia have been eminently satisfactory, it is in the large ventral hernias that we have been most impressed with the value of the method. After the sac has been removed or pushed back, the muscular and aponeurotic edges of the ring are cleared sufficiently for the insertion of the sutures. In our early operations, we simply imitated the well-known overlapping methods, separating the various layers of the abdominal wall from one another and overlapping them when it was possible. We soon found, however, that this very decidedly weakened the boundaries of the defect and that, whenever it was necessary to draw them together under strong tension, the stitches

were much more apt to tear holes than when they were left undisturbed as a solid mat. When we fully appreciated that the sutures themselves could be depended upon to remain unchanged for all time, we abandoned all dissection of the edges of the ring and contented ourselves with simple edge-to-edge apposition in the suturing. When it is seen that the boundaries of the ring can be drawn together without too great increase of the intra-abdominal pressure, the living suture is anchored at one end of the wound, and the opening is closed with a through-and-through running stitch (Fig. 7). The only care to be taken is to see that the needle always passes through tough aponeurotic tissue, so that all possibility of the stitches tearing out is eliminated. The opening

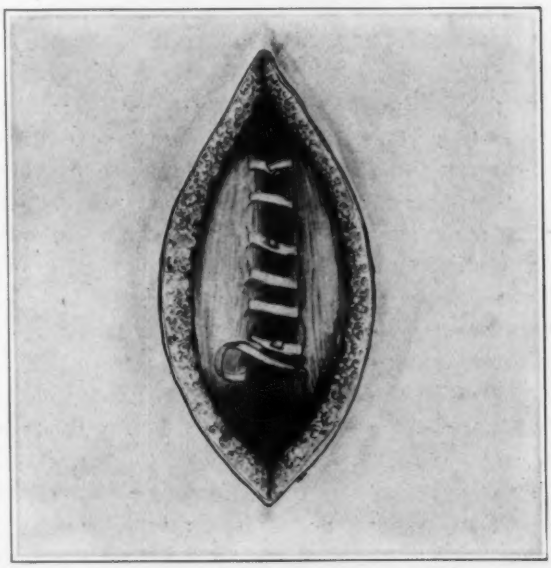

Fig. 7.—Repair of ventral hernia; insertion of first row of sutures, bringing the edges of hernial ring as nearly together as possible.

is further supported by a second layer of sutures superimposed on the first, which gets its grip of the abdominal wall at a distance of an inch from the hernial ring, and the suture crosses and recrosses the former opening much as a lace in a shoe (Fig. 8). This row of sutures is not drawn tighter than is necessary to make the strips of fascia lie flat, so that no inversion of the wound occurs. When it is seen that the large size of the hernial ring makes it impossible to draw the edges together, which is frequently the case in extensive shell wounds of the abdominal wall or when the edges can only be drawn together under very severe tension by means of Kocher clamps, we abandon all idea of bringing them into actual contact and set about repairing the defect by other

means. This may be accomplished by filling in the opening exactly as a woman darns a hole in a sock. The sutures are drawn sufficiently taut to preclude unnecessary waste of material, but under no circumstances so taut as to produce undue compression of the abdominal contents.

This method of suture has proven highly satisfactory in a series of cases extending over four years. Some of the hernias were enormous, and the surgeons who saw them considered them incurable. We are particularly proud of the case of a soldier who came in with a hernia half the size of a football, resulting from a shell wound, which had destroyed the upper half of the right rectus muscle and a large area of the abdominal aponeurosis. His abdominal wall in this region

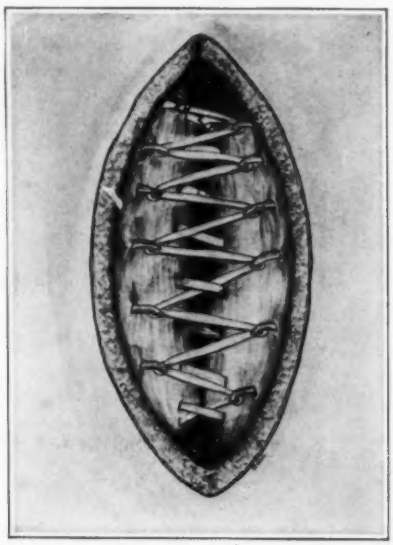

Fig. 8.—Repair of ventral hernia; insertion of second row of sutures, fixed at points some distance from the edges of the hernial ring.

consisted of an exceedingly thin scar through which peristaltic movements of the intestines were plainly visible. The muscles in the outer edge of the hernial ring had retracted to beyond the anterior axillary line, leaving an opening about 7 inches by 5. This hernia was closed about three years ago by the darning method described, and the man has been working as a laborer ever since, without disability. He has very little subcutaneous fat, and in that portion of the wound in which the edges of the ring could not be drawn together, the sutures can still be felt, something like the ridges on a washboard.

More recently, we have been trying out in very wide hernias another method, which removes the possibility of a protrusion of a sac through

the chinks that remain between the sutures. In this operation, two patches of fascia lata are removed from the thigh and trimmed to the right shape to fill the gap in the abdominal wall. An ample margin is left for an overlap. The two pieces of fascia are then laid in the wound, one on top of the other, in such a way that the fibers of the two layers cross one another obliquely, as they do in the various layers of the abdominal aponeurosis. The transplants are then sewed into position by two rows of living sutures running all the way around the edges of the opening. In this operation one can recognize a certain resemblance to the old-fashioned patch on the seat of trousers.

The removal of the fascial sutures from the thigh results in no disability. As a rule, four strips of fascia are all that are required, and following the operation there is no difficulty in closing the opening with catgut suture. In cases, however, where patches were required or where so many sutures were needed that the opening in the fascia lata could not be closed, the patients made no mention of their thighs, and no deformity or disability resulted. The wound in the thigh does not lengthen the period of stay in bed.

At present we have records of approximately one hundred hernias of all varieties, which have been closed by means of living sutures. In this series, there have been, up to the present, no recurrences. Three cases have suppurated, but, fortunately, the suture was not extruded and no ill effects have ensued. This is probably due more to good luck than to any virtue in the method of suture used. Among these hernias were many which we feel sure could not have been cured by the ordinary methods of operation, and we are therefore very hopeful that the use of living sutures will make a decided reduction in the percentage of recurrences and will considerably widen the field in which operative treatment is applicable.

The method is recommended for the following conditions: (1) direct inguinal hernia; (2) oblique inguinal hernia in patients at or beyond middle age, particularly where the canal has lost its obliquity and where the abdomen is becoming pendulous; (3) femoral hernia, in which the suture is used to close the entrance of the femoral ring through an inguinal incision; (4) all forms of recurred hernia; (5) all ventral hernias in which there is any doubt in the surgeon's mind as to the permanence of closure by the ordinary methods, and (6) ventral hernia, in which closure can be obtained only with disquieting increase of intra-abdominal pressure.

# STUDIES IN ASEPTIC INTESTINAL ANASTOMOSIS

## II. AN ASEPTIC METHOD OF END-TO-END INTESTINAL ANASTOMOSIS BY THE INVAGINATION OF THE LIGATED BLIND ENDS OF THE INTESTINES TO FORM DIGESTIBLE BOLI, RELEASE OF WHICH REESTABLISHES THE CONTINUITY OF THE INTESTINAL LUMEN *

WALTER C. BURKET, M.D.
EVANSTON, ILL.

The present method of aseptic intestinal anastomosis was developed to eliminate the objectionable use of any special instrument or mechanical device which must be either withdrawn through the line of suture before the final closure or passed out of the intestines by peristalsis or otherwise. Also complications, such as postoperative adhesions and infection, are still important problems consequent to the open suture of the intestine. The following, quoted from Dr. William Stewart Halsted,[1] emphasizes this factor: "Soiling unquestionably contributes to the mortality attending the circular suture, particularly of the large bowel, but how important this factor is cannot be definitely determined until it shall have been completely eliminated."

### HISTORICAL REVIEW

Many mechanical devices, knives, clamps, buttons, release sutures, etc., have been described from time to time. These have been enumerated in an article that was published by myself in collaboration with Dr. William B. McClure[2] in 1922. Since that time, papers have appeared treating either of new mechanical devices or of removable and release-string sutures, by Collins,[3] Miller and Andrus,[4] Bidgood,[5] Kerr,[6]

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* From the Otho S. A. Sprague Memorial Institute Laboratory of the Children's Memorial Hospital, Chicago.

1. Halsted, W. S.: A Bulkhead Suture of the Intestine, J. Exper. M. **15**: 216-224, 1912.

2. Burket, W. C., and McClure, Wm. B.: An Aseptic Method of Intestinal Anastomosis: An Experimental Study, Surg., Gynec. & Obst. **35**:816-819 (Dec.) 1922; abstr. J. A. M. A. **80**:62-63 (Jan. 6) 1923; abstr. Surg., Gynec. & Obst. **36**: 217, 1923.

3. Collins, F. K.: Aseptic Resection of Intestine by Use of Special Application of Removable Looped Ligature, Ann. Surg. **76**:739-744 (Dec.) 1922.

4. Miller, R. T., Jr., and Andrus, W. D. W.: Experimental Surgery of the Thoracic Esophagus, Bull. Johns Hopkins Hosp. **34**:109-114 (April) 1923.

5. Bidgood, C. Y.: Blind End-to-End Anastomosis of the Sigmoid; a Modified Halsted Operation; Presentation of a New Instrument; Bull. Johns Hopkins Hosp. **34**:197-199 (June) 1923.

6. Kerr, H. H.: Intestinal Surgery, J. A. M. A. **81**:641-647 (Aug. 25) 1923.

Horine,[7] Highsmith [8] and Trueblood.[9] It would seem that the removal of any device or suture through the line of anastomosis in order to open the intestinal lumen is a break in aseptic technic and should be limited to cases in which operative difficulties prevent any other method.

An anastomotic opening reestablished relatively promptly seems necessary in the small intestine in order to be compatible with life. Delay in the reformation of the lumen in the large intestine for a short period may not cause death in some animals, but it is certainly not desirable. In the cat, temporary obstruction of the large intestine for twenty-four hours results in death. Hence, methods that depend on the sloughing of a cauterized or crushed intestinal diaphragm or the absorption of a suture to permit the lumen to be reestablished are, in the author's opinion, not suitable for application to the human subject.

The development of the present method was begun in 1916, and various phases of the problem were studied in a series of cats in 1917 until war service interfered with work. Subsequently, the procedure has been greatly improved and studied on dogs. Briefly, the operation (Figs. 1-5) consisted in invagination of the ligated intestine ends (Fig. 2), like an appendix stump, under ligatures to form digestible release boli of the intestinal wall itself, redivision with the actual cautery of the intestine distal to the ligature (Fig. 3), complete circular suture of the abutted blind intestine ends (Fig. 4), and release of the digestible boli to reestablish the intestinal lumen. This operation includes phases of Dr. William S. Halsted's bulkhead circular suture method [1] but, in my opinion, solves the problem of the bolus in a very simple manner, which Halsted's method does not do. Dr. Halsted [10] refers to the problem of crushed or cauterized diaphragms but not to a bolus made from the intestinal wall itself:

> Did it suffice merely to sew together the abutted ends of the gut, each having been treated after the manner of an appendix stump, the problem would seem to be solved. The intestinal wall having been crushed and reduced to its submucosa, firmly ligated and divided with the Paquelin's cautery, the division of the gut is aseptically accomplished; the two ends can now be abutted and sutured without manifest flaw in the technic, but a double diaphragm remains

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7. Horine, C. F.: Aseptic Technique for the Resection of Intestine; Blind End-to-End Anastomosis with the Release of Purse-String Suture After the Anastomosis Is Complete, Ann. Surg. **76**:745 (Dec.) 1922; ibid., Surg., Gynec. & Obst. **79**:100-101, 1924.

8. Highsmith, E. D.: Aseptic Resection of Intestine, Surg., Gynec. & Obst. **36**:271-273 (Feb.) 1923.

9. Trueblood, D. V.: End-to-End Intestinal Anastomosis, An Experimental Study, Northwest Med. **22**:27-29 (Jan.) 1923.

10. Halsted, W. S.: End-to-End Suture of the Intestine by a Bulkhead Method, Tr. Am. Surg. Assn., Philadelphia **28**:256-261, 1910.

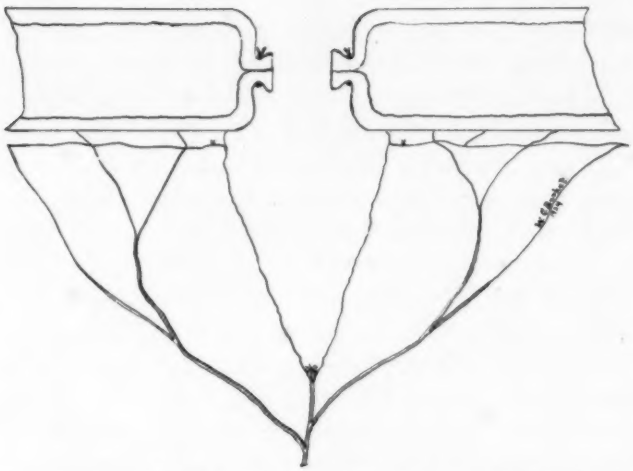

Fig. 1.—Diagrammatic longitudinal section of the small intestine; mesenteric blood supply ligated; intestine resected with actual cautery; ligated stumps ready for invagination.

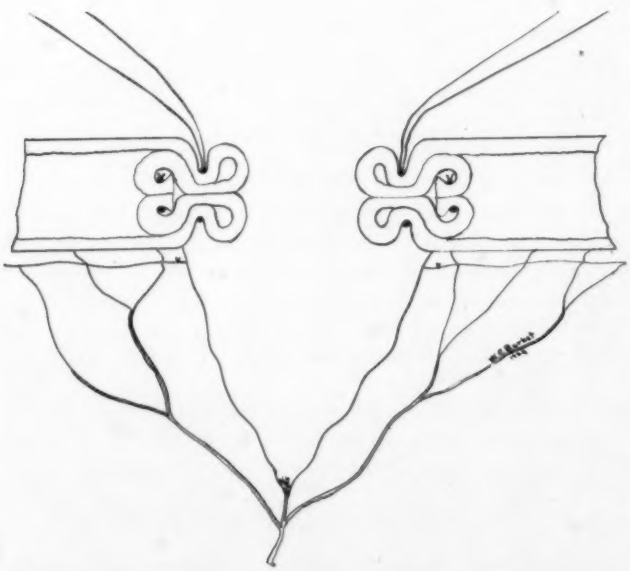

Fig. 2.—Diagrammatic longitudinal section of the small intestine; cauterized stumps invaginated under a ligature with the ends left long.

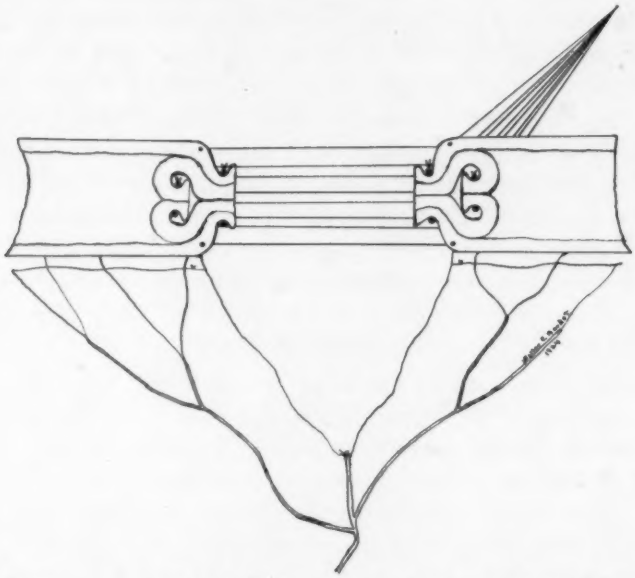

Fig. 3.—Diagrammatic longitudinal section of the small intestine; intestine distal to ligatures cauterized off; digestible release boli completely formed; presection sutures from one half of the circumference of the intestine drawn taut.

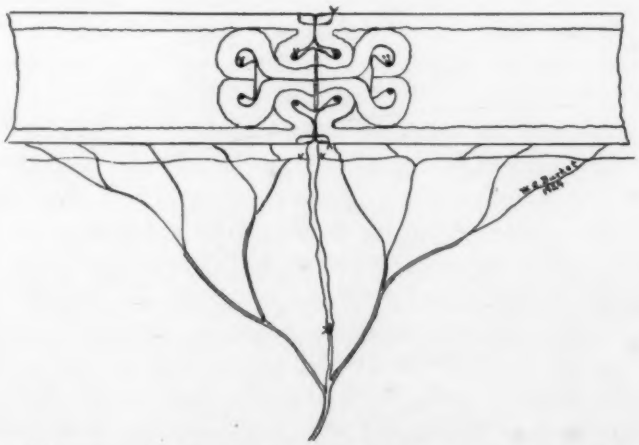

Fig. 4.—Diagrammatic longitudinal section of the small intestine; circular suture of abutted intestine ends completed; digestible boli ready to be released.

which would impede for a long time the passage of intestinal contents even if the ligatures employed in the tying off of the gut could be relied upon to melt away or disappear in a desired period.

Dr. Halsted and Dr. Gatch [11] searched widely for a suitable bolus, and Dr. Grey [12] used blood fibrin cones. Suffice it to say that a satisfactory bolus was never obtained. Another radical difference from the bulkhead method is that the cone-shaped boli occupy a reversed position; i. e., in the author's method the base of the cone is toward the intestinal lumen. The bulkhead method requires a specially prepared and sterilized device for the bolus; my method of operation requires none.

## OPERATIVE METHOD

The aseptic method (Figs. 1-5) of intestinal anastomosis is as follows: the mesenteric blood supply of the bowel to be resected and anastomosed is ligated, and the mesentery is trimmed away from the intestinal wall with scissors. The isolated intestine (Fig. 1) is resected between ligatures of heavy silk with the actual cautery knife. Each intestine end (Fig. 2) is invaginated, like an appendix stump, with the aid of a clamp and held in place by a firmly tied heavy silk ligature (catgut may be used if preferred), the ends of which are left long to serve as a handle to steady and control the intestine so that direct handling of the intestine itself is avoided considerably. Between the intestine ends (Fig. 3) a complete row of presection, interrupted, fine black silk anastomosing mattress sutures is placed proximal to the ligatures that hold the invaginated stumps, and at the location where the blood supply to the intestinal wall is perfectly maintained. These sutures are not tied; each one has a clamp attached to facilitate identification, and the sutures are kept together in two groups corresponding to the respective halves of the circumference of the intestine, so that the intestine ends may be brought up between the two groups of sutures. The sutures in the mesenteric surface of the intestine are placed first, in order to aid in the accuracy of their insertion. The operative field is protected with wet pads which are fitted snugly around the intestine at the site of the ligatures, and the portion of the intestine that protrudes distal to the ligature is cauterized off (Fig. 3) with the actual cautery knife. This latter procedure is much easier, when plenty of bowel is available, by placing on the intestine protruding distal to the ligature, a second ligature so that cauterization may be carried out between ligatures. The distal segment cauterized off is

11. Gatch, W. D.: Aseptic Intestinal Anastomosis; An Experimental Study, J. A. M. A. **59**:185 (July 20) 1912.

12. Grey, E. G.: Studies on the Aseptic End-to-End Anastomosis of the Intestine, Bull. Johns Hopkins Hosp. **29**:267-271 (Dec.) 1918.

discarded. This cauterization makes ready for release the digestible boli which consist of the original invaginated stumps with their contiguous intestinal wall that protrudes from under the ligature (Fig. 3). These cone-shaped boli are held in place by the ligatures and cannot force themselves outward because the base of the cone is directed toward the intestinal lumen. After both intestine ends have been similarly redivided with the actual cautery, distal to their ligatures, the presection mattress sutures are drawn up and tied so that they completely anastomose together the abutted, blind intestine ends (Fig. 4). A second row of anastomosing interrupted mattress sutures, alternating with the sutures of the first row, is now placed and tied at the time of insertion. When the sutures have all been tied, gentle pressure is applied

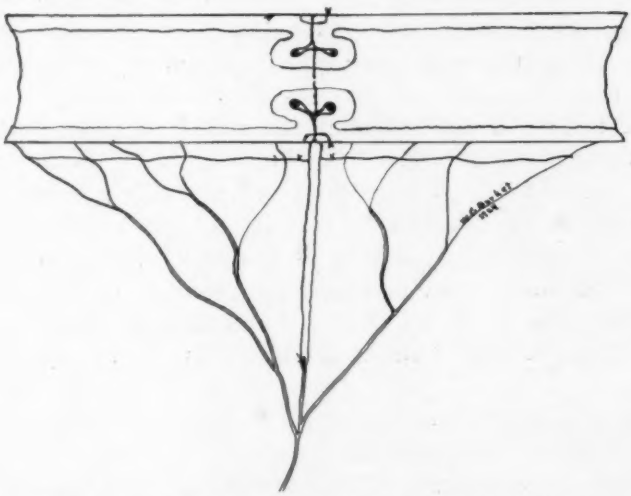

Fig. 5.—Diagrammatic longitudinal section of small intestine; continuity of the intestinal lumen reestablished.

directly over the line of anastomosis and consequently falls on the apexes of both cone-shaped boli which readily slip free in opposite directions within the bowel; thus is promptly reestablished the intestinal lumen (Fig. 5) which has not been opened at any time during the operation. The rent in the mesentery of the small intestine is approximated with a continuous suture. The mesentery of the large bowel usually needs no closure.

## STUDY OF THE ASEPTIC METHOD

I have employed the foregoing method by aseptic operation on six dogs under ether anesthesia. The dogs were of various sizes and ages. Some of the animals have been operated on several times for intestinal anastomosis, including both the large and small intestines. The

anastomoses, verified at a second operation, have been seen to be neatly healed without dilatation or stricture of the intestine and, at times, without the slightest adhesion. The dogs are all living and well after a period of some months. They are well nourished. Convalescence was without symptoms and prompt; all wounds of both the large and small intestine healed by first intention following primary closure. Soiling from the intestinal lumen did not occur. The operations were performed without any assistant other than an anesthetist. Fine black silk sutures were used through the entire operation. No catgut was employed. The technic is so arranged that very little handling of the intestine occurs.

## CONCLUSIONS

1. The method is a simple, clear-cut, operative procedure that makes end-to-end intestinal anastomosis, especially of the large bowel, a much safer undertaking than the open method, certainly from the standpoint of soiling and infection.

2. The method is applicable to both small and large intestines in an animal of any size or age.

3. No special mechanical device or instrument is required.

4. No material is withdrawn nor is any operative procedure carried on through the line of suture in order to reestablish the lumen.

5. No instrument or nondigestible mass, such as the button used in intestinal anastomosis, is left in situ to interfere with healing or anything that must be passed out of the intestines either by peristalsis or otherwise.

6. The anastomosing sutures are all tied before the intestinal lumen is reestablished.

7. Asepsis is dependent on the effectiveness of the actual cautery and the skill with which sutures are placed in the submucosa without penetrating the mucosa.

8. A method is available which will permit the study, free from the factor of infection from soiling, of various problems relating to intestinal anastomosis, such as the problem of postoperative adhesions, the selection of the most suitable number and kind of sutures, etc.

It is intended at a later date to present further studies of aseptic intestinal anastomosis.

# ECHINOCOCCUS CYST IN THE SCAPULAR AREA

## REPORT OF A CASE *

MINAS JOANNIDES, M.D.

AND

WILLIAM A. RILEY, PH.D.

MINNEAPOLIS

This case is reported because of the uncommon location of the cyst, and because it gives strong evidence that the infection may be carried through the general circulation to various parts of the body.

### REPORT OF CASE

*History.*—Mrs. M. K., aged 45, a Greek housewife, came to one of us July 26, 1923, complaining of a painful lump over the left scapula. This mass had been noticed ten days previously, and its presence was attributed by the patient to her having "caught cold." At that time a smaller mass appeared at the region of the right pole of the thyroid gland.

The family history was negative with the exception that her father also had had a lump on his left shoulder, which had disappeared spontaneously. Her husband and two children gave no history suggestive of echinococcus disease.

The patient came to America in 1920. Shortly after arriving in this country she had noticed a mass the size of a hen's egg over her right shoulder. This mass disappeared after one or two rubs with liniment. Otherwise, the past history was negative. The evening of July 16, while the patient was out in the park she had a chill, and afterwards had "felt hot." Then she noticed pain over the left scapula and in the neck. She rubbed herself with liniment but did not obtain much relief. The mass over the scapula rapidly grew larger and more painful. The one at the region of the thyroid did not enlarge, and produced only very slight pain.

*Examination.*—When she came under observation there was a mass over the left scapula. This was subcutaneous, and was loosely attached to the scapula and the chest wall. The skin at this area was not attached to the mass, which was approximately 10 cm. in diameter, and about 4 to 5 cm. above the level of the skin. Fluctuation could be made out with some difficulty because the mass was quite tense. The mass in the right pole of the thyroid gland was approximately 5 cm. in diameter and was continuous with the upper right pole of the thyroid. It was slightly hard, but not tender. The rest of the examination was negative.

*Diagnosis.*—On account of the slight rise of temperature (99 F.) and the severe pain that was present, the formation of an abscess was suspected. An incision, one inch in length, was made under gas anesthesia. After the skin and superficial fascia were incised, a tough capsule was encountered. When,

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* From the departments of Surgery and Entomology, University of Minnesota, Minneapolis.

however, the capsule was nicked, thick yellow pus appeared under pressure. The capsule was then incised and along with the pus a large number of grape-like cysts appeared. These varied from a few millimeters to over two centimeters in diameter (Fig. 1). The microscopic examination of the cysts clinched the diagnosis of echinococcus.

*Operation.*—The patient was sent to St. Mary's Hospital, where, July 28, under ether anesthesia, the incision was enlarged and the cyst removed, care being taken to remove also the fibrous wall of the cyst. Surgical solution of chlorinated soda (Dakin's solution) was applied to the exposed surfaces, and the wound was sutured with tubes placed at the upper and lower portions of the incision. Before working on the scapular cyst, an attempt was made to remove the mass at the region of the thyroid gland. After a collar incision was made, and we were beginning to separate the sternothyroid muscles, the anesthetist informed us that the patient was taking the anesthetic very badly. In view of the fact that the scapular cyst was open, and toxic material could be

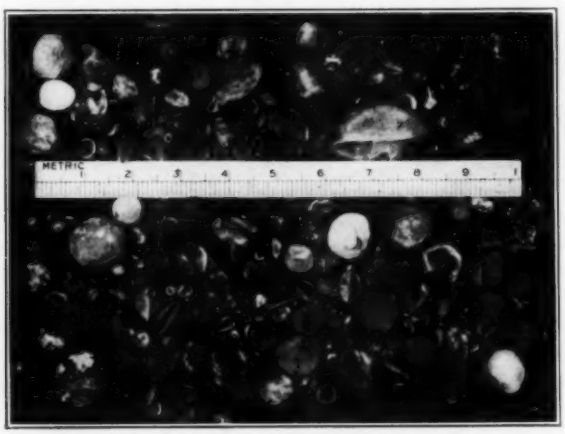


Fig. 1.—The grape-like cysts obtained on incision of capsule.

absorbed by the raw surfaces, and because of the poor condition of the patient under the anesthetic, we gave up the plan of removing the thyroid mass, made a hurried closure, and, after turning the patient on her abdomen, we removed the scapular cyst.

*Course.*—The postoperative course was uneventful. The patient had a slight rise of temperature for five days. The scapular wound was treated with Dakin irrigations. The neck wound healed by first intention. The patient was discharged from the hospital August 7, and the wound had completely healed by August 24. Curiously enough, the mass connected with the thyroid gland had completely disappeared when she was discharged as cured. Six months after the operation the patient was free from any symptoms.

A considerable number of the daughter cysts were minutely examined, and the remainder less critically for the presence of scolices and hooks. None was found, and neither was there any trace of them in the fluid content of the cyst. This acephalous condition has been noted frequently in man; in fact, it is only rarely that the hooks and heads are noted in human cases.

The echinococcus fluid is usually limpid, of a light yellow color and sterile. In the case under consideration there was an abundance of pus present, the infection being responsible for the painful condition and for the rise in temperature which had occurred.

In spite of the voluminous literature on echinococcus disease, there are very few references to cysts located in the scapular region.

As is well known, the parasite is most commonly found in the liver. Thus, Teichmann,[1] in his analysis of 2,452 cases records 1140, or 46.4 per cent., for the liver; 214, or 8.76 per cent., were located in the lungs, and next in order were the female genital organs with 156, or 6.3 per cent. of the cases. Teichmann's tabulation places next to the liver cases those of "superficial organs" with a total of 337 or 13.7 per cent. However, in this grouping he includes those of the eye, mouth cavity, thyroid, larynx and of the skin, muscle and subcutaneous tissue in general; 21, or 0.85 per cent. of the total, were in the anterior thoracic, lateral, or posterior thoracic wall.

It is interesting to find that the first definitely recognized case of echinococcus in man, described by Bremser in 1821 [2], concerned a cyst the size of a hen's egg, which had been removed from the subclavicular region of a woman.

Davaine [3] cites the observations of Andral, 1829, on a patient who had in the middle of his scapula a tumor of uncertain nature, from which on incision there escaped a large number of acephalocysts. The patient died as a result of the operation, and on examination there was found a group of the daughter cysts ("paquet de ces entozoaires") in the infraspinous fossa and another in the subscapular fossa. The two groups communicated by a neck passing through the scapula, not far from the spine.

Within recent years, Kothary [4] has reported the case of a Mohammedan woman in India who came for treatment for a hydatid cyst on the back, of seven years' duration. In the beginning there was a small tumor about the size of a walnut over the back below the left scapula. After four years, this began to increase in size and became somewhat painful. When she was admitted to the hospital the tumor was the size of a cocoanut. It was movable, but the skin over it was adherent. There was no fever and no wasting, and the general condition was satis-

1. Teichmann, Curt: Zur Localisation des Echinococcus im menschlichen Körper. Diss. Halle-Wittenberg, 1898, p. 51.

2. Bremser, J. G.: Etwas über Echinococcus hominis Rud, Deutsches Arch. f. d. Physiol. **6**:292-302, 1820.

3. Davaine, C.: Traité des entozoaires et des maladies vermineuses de l'homme et des animaux domestiques. Ed. 3, Paris, Bailliere and Son, **132**:1003, 1877.

4. Kothary, P. T.: Hydatid Disease in Kathiawar, Indian Med. Gaz. **52**: 273-275, 1917.

factory. It was believed to be a sebaceous cyst. An oval incision was made over the cyst; after the skin was retracted the wall was dissected off. "In this process the cyst burst, and, to our surprise, discharged some watery fluid together with daughter cysts of a hydatid cyst." The whole wall was removed and the skin sutured, keeping a little drain. The after treatment and progress were uneventful, and the patient was discharged cured after a fortnight or so.

Although cases of hydatid disease are sufficiently uncommon and striking enough to arouse interest, the disease is not to be regarded as especially rare. The cysts were known to the ancients. As was true of other larval tapeworms, they were regarded as inflammatory products, or abscesses, down to the time of Pallas.[5] This keen observer noted the heads of tapeworms in the cysts, while Goeze [6] recognized them as true tapeworm heads and described the suckers and hooks of animal and human echinococci. Discussing his findings, he states, "I only wish that I could throw more light on the method of origin of these bladders, and on the economy of the many thousand individual worms associated together in the single bladder. Do they become larger? Do they divide? Does each one form its own habitation, or where do they remain?"

In 1808, Rudolphi [7] found the adult worms in the intestine of a house dog in large numbers and, in keeping with current theories regarding the origin of tapeworms, he considered them as spontaneously produced from the intestinal villi. It remained for von Siebold [8] and for Küchenmeister [9] to rear adults by feeding dogs echinococcus cysts from cattle. In 1862, Naunyn [10] and later Krabbe [11] and Finsen obtained the adults in dogs that had been fed cysts from man.

## THE PARASITE

Taenia echinococcus is one of the smallest of the numerous species of tapeworms of mammals, measuring from 2.5 to 6 mm. in length and 0.6 mm. in breadth. The head measures about three-fourths millimeter

5. Pallas, P. S. (cited by R. Leuckart): The Parasites of Man, Edinburgh, Pentland, 1886, p. 583.

6. Goeze, J. A. C.: Versuch einer Naturgeschichte der Eingewedewrmer thierischer Körper, Blankenburg, **11**:471, 1782.

7. Rudolphi, C. A.: Entozoorum sive vermium intestinalium historia naturalis, Amstelodami, 1808, p. 411.

8. von Siebold, C. T. E.: Ueber die Verwandlung der Echinococcus—Brut in Taenien, Ztschr. f. wissensch. Zool. **5**:409-424, 1853.

9. Küchenmeister, Fr.: Cited in his Die in und an eubner. dem Körper des lebenden Menschen vorkommenden Parasiten, Leipzig, 1855, p. 141.

10. Naunyn, B.: De echinococci evolutione, Diss., Berlin, 1862, pp. 1-35.

11. Krabbe, H.: De islandske Echinokokker, Virchows Arch. f. Anat. und Phys. **27**:225-239, 1863.

in breadth, has four suckers, and a double row of hooks of various dimensions. Generally there are 30 to 50 hooklets on the rostellum. The neck is very short, and the body consists of three to four proglottids. The terminal one, when mature, contains 400 to 800 eggs with their hexacanth embryos. Taenia echinococcus is found in the dog and closely related carnivores such as the jackal or the wolf. On the other hand, there are some forty animals reported as hosts for the larval form. Among the intermediate hosts the following may be mentioned, man, hog, cattle, sheep, goat, camel, dromedary, horse, deer, donkey, rabbit, kangaroo, giraffe, and various rodents. It is especially common in the hog. The ovum, measuring 30 to 40 microns in diameter, contains the embryo. Leuckart has shown that after it is ingested by the intermediate host, the membrane of the ovum is dissolved by the gastric juice in 4 to 5 hours. The liberated embryo is then mixed with the stomach contents and carried to the intestine and there by means of its hooks attaches itself to the intestinal mucosa. The embryo then penetrates the intestinal wall and after reaching the capillaries it is carried in the circulation to the various parts of the body, giving rise to the hydatid cyst. Devé [12] produced experimentally metastatic cysts in the brain of the rabbit by injecting the carotid artery with scolices. There are some observers who contend that metastases occur by actual migration of the embryo to the various organs. The wall of the cyst is made up of an external laminated layer and of an internal germinative or parenchymatous layer. The inner layer gives rise to daughter capsules and within these scolices develop. Leuckart's studies have shown that this process is very slow, from five to six months being required for growth and maturing of the cysts. Granddaughter cysts may develop from the daughter cysts. Thus one ovum may give rise to several hundreds of adult worms. A thick fibrous layer is produced by the host around the cyst.

### DISTRIBUTION

Like its primary host, the dog, this parasite is cosmopolitan in its distribution. Iceland has long served as the "horrible example" of the relation of the disease in man to close association with the primary host. There, where this animal plays so important a rôle in the life of the population, it was stated by Krabbe [11] that there was one dog to every three persons and that 28 per cent. of them harbored the mature worm. Under such conditions it is not surprising that the disease was very prevalent in man. Thorstensen [13] goes so far as to

12. Devé, F.: Echinococcose cérebrale metastatique experimentale, Compt. rend. Soc. de biol. **133**:511-512, 1920.

13. Thorstensen: Hydatiden und Entozoen in der Leber, Arch. d. k. dän. Gesundheitscool, 1848. (Cited by Küchenmeister, 1855.)

say that every seventh person harbored the cysts, and that in some localities the proportion rose to one in three. Such astounding figures, though often quoted, are derived from the early literature on the subject and may well be questioned. Hence it is significant to note that Magnusson [14] states: "The fact that for some years past practically nothing has appeared concerning echinococcus in Iceland, unfortunately does not mean . . . that the disease has died out here or even that it has become rare. In each of the some twenty years that I have practiced on the island I have seen many cases. . . . Finsen estimated the number of echinococcus patients at 1:40 or 1:50 of the entire population; Jonassen at 1:61." Magnusson tabulates for the period of fifteen years preceding 1912 some 1,650 cases, or 110 per year for Iceland. In the leprosy hospital near Reykjavik, twenty-six cases were found in eighty-four necropsies.

In certain regions of Australia, particularly in Tasmania and the province of Victoria, the parasite is almost equally prevalent. Thomas [15] makes the surprising statement that 43 per cent. of the dogs harbor the worm.

In recent years attention has been directed to the extreme frequency of the disease in South America, particularly in the Argentine Republic and in Uruguay. So serious a condition developed in Argentina that, as noted by Blanchard,[16] the minister of agriculture appointed a commission charged with the study of prophylaxis against the disease. In both countries, placards warning the public against the danger have been widely distributed.

Coming closer home, the echinococcus cysts are by no means rare in domesticated animals in this country. Dr. B. H. Ransom [17] called attention to the fact that the federal inspectors were finding them in alarming numbers in meat from parts of Virginia, Oklahoma, and Arkansas. Dr. Stiles [18] states, "Wheeler records 117 cases of liver echinococcus in 2,000 hogs examined at New Orleans." Dr. G. Dikmans,[19] who has made many examinations of hogs at the abattoirs at Baton Rouge, Louisiana, informs one of us (Dr. Riley) that he has found about the same proportion as noted by Dr. Wheeler.

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14. Magnusson, G.: 214 Echinokokken—operationen. Beitrag zur Pathologie und Therapie der Echinokokkenkrankheit, Arch. f. klin. Chir. **100**:293-381, 1913.

15. Thomas, D.: Hydatid Disease in Australia, London, Lancet, March 1, 1879, pp. 297-298.

16. Blanchard, R.: Nouvea cas de Dipylidium caninum á Paris, Archiv. de Parasitologie **13**:243-250. 1909.

17. Ransom, B. H.: Report of the Chief of the Bureau of Animal Industry, U. S. Dept. Agriculture, 1915.

18. Stiles, C. W.: In Osler and McCrae's "Modern Medicine," Philadelphia, Lea & Febiger, **2**:267-268, 1913.

19. Personal communication.

Lyon [20] presented a detailed review of echinococcus disease of man in North America. He brought together 241 cases, twenty of which were museum specimens. Seventy-four per cent. of the patients were middle-aged persons, and 68 per cent. of these were males. Of those whose nativity was known, 91 per cent. were foreign born. Of these the greatest number, fifty-eight, were immigrants from Iceland, and twenty-three were from Germany.

We had completed a large part of the record to date when we found that Magath [21] had not only added sixty-eight cases from the literature, but had recorded twenty-five unreported cases from the Mayo Clinic, thus making a grand total of 334 cases for North America.

Of these 334 patients, the nativity of 236 was known. Eleven of them were born in the United States, and four in Canada. It is interesting to note that while there were no cases reported among Greeks in the United States prior to 1902, there have been seventeen (now eighteen) reported since then. Magath points out that this is correlated with a greatly increased immigration from Greece in recent years.

In view of this prevalence of the cysts, or larval stages, of the echinococcus in the United States, it is obvious that the adult worm must be fairly common in dogs in many sections of the country. Curiously enough, aside from experimental infections, it seems to have been reported but once for the United States. In a list of the parasites infesting the domesticated animals and man in the United States, Cooper Curtice [22] makes the statement, "*Taenia echinococcus* von Siebold, very minute, found but once in a dog killed at the Washington, D. C., pound." This failure to note the occurrence of the worm is in part due to its small size, but more especially to the fact that in spite of the bearing on public health problems, we have very few attempts at a census of the parasites of the dog in this country. Practically all the lists available relate to northern localities. It is very desirable that careful studies be made in various southern states. In this connection, it should be emphasized that caution must be exercised in handling possible carriers of the echinococcus tapeworm, since the embryos are so readily transmissible to man.

## DIAGNOSIS

With the exception of hydatid fremitus, the symptoms depend entirely on the location of the cyst. Frequently, the cysts do not pro-

20. Lyon, G. P.: A Review of Echinococcus Disease in North America, Am. J. M. Sc., New Series **123**:124-141, 1902.

21. Magath, T. B.: Echinococcus Disease: Etiology and Laboratory Aids to Diagnosis, Med. Clin. of No. America **5**:549-71, 1921.

22. Curtice, Cooper: Parasites: Being a List of Those Infesting the Domesticated Animals and Man in the United States, Jour. Comp. Med. and Vet. Archives, New York **13**:223-236, 1892.

duce any symptoms until secondary infection occurs, and then one notices the picture of an acute infection. Abdominal hydatids sometimes rupture spontaneously and cause death from the absorption of very toxic substances found in the fluid. In the lungs, rupture may occur into the bronchus. Daughter cysts or hooklets are then expectorated and frequently discovery of such cysts or hooklets leads to the diagnosis of hydatid cyst of the lungs.

Coincident with rupture of hydatid cyst, one often finds urticaria. This is due to sensitization of the host against hydatid cyst antigen. It is the recognition of the presence of a specific antigen in the hydatid fluid that has led such men as Ghedini,[23] Weinberg and his associates,[24] and recently, Fairley [25] and Magath [21] to study the biologic reactions in hydatid disease. Some have found the roentgen ray (Halahan [26]) of great value in the diagnosis of hydatid cyst of the lungs and pleura. An absolute diagnosis is only made after recognition of the daughter cysts or hooklets.

## TREATMENT

The treatment of hydatid disease is admittedly surgical. Excision of the cyst frequently cures a patient completely. Recurrences after the first operation have been reported by Halstead [27] and Norburn.[28] Albo [29] states that drugs such as arsphenamin and neo-arsphenamin are regarded as having no particular value. Halahan [26] regards aspiration of hydatid cysts as a dangerous procedure because of possible absorption of toxic cyst products. O'Connor,[30] on the other hand, considers aspiration of cysts of the lungs and pleura of great value. Marsupialization is a means of treating abdominal cysts which are difficult or impossible to excise. In our case, excision of the cyst resulted in a cure and no recurrence was noticed six months after operation.

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23. Ghedini: Richerche sul Siero del Sangue, Gazz. d. Ospitali, Milan **27**: 1616, 1906; and **28**:53, 1907.

24. Weinberg and Parvu: Reaction de Bordet—Gengon dans les Helminthiasis, Compt. rend. Soc. de biol., Paris **65**:298, 1908. Weinberg and Vieillard: Echinocoquose de Chameau, Bull. Soc. Centrale de Med. veterinaire, Paris, 1909. Weinberg: In Kolle and Wassermann's Handbuch der Pathogenen Mikro-organen, 1913.

25. Fairley, H.: Researches on Complement Fixation Reaction in Hydatid Disease, Quart. J. Med. **15**:244 (April) 1922.

26. Halahan, R.: Echinococcus Cyst of the Lungs and Pleura, Surg., Gynec. & Obst. **36**:354 (March) 1923.

27. Halstead, A. E.: Echinococcus Cyst of the Liver, S. Clinics of N. America **3**:973-983 (Aug.) 1923.

28. Norburn, C. S.: Recurrence in a Case of Hydatid Disease, U. S. Nav. M. Bull. **16**:288 (Feb.) 1922.

29. Albo, M.: Hydatid Cysts of the Pancreas, Surg. Gynec. & Obst. **34**:739. 1922.

30. O'Connor, J.: Surgical Treatment of Hepatic Abscess and Hydatid Cysts Evacuating Via Bronchus, Ann. of Surg. **76**:591 (Nov.) 1922.

# SARCOMA OF BONE: AN ANALYSIS OF FIFTY CASES

C. J. MacGUIRE, Jr., M.D.
AND
J. E. McWHORTER, M.D.
NEW YORK

The cases in this series were collected from the Presbyterian, New York, and Bellevue Hospitals, New York, and include all those cases since 1909 in which the diagnosis of sarcoma of the bone could be definitely confirmed. Recent analyses along similar lines by Greenough, Simmons and Harner [1] of Boston, Morton and Duffy [2] of New Haven, and Meyerding [3] of Rochester have demonstrated the value of supplementing the work of the central registry for bone tumors in Boston by recording in local group presentations those clinical features and personal reactions which are inevitably lost, to a large degree, in a national statistical bureau.

It is our aim to bring out the difficulties that surround the accurate study of these neoplasms, particularly those relating to their diagnosis, prognosis, surgical treatment and classification. A general review of the subject has not been attempted, as this academic field has already been covered by Bloodgood,[4] Ewing,[5] and many others.

Up to this time, there have been no cases included which lacked a follow-up or a microscopic section. In spite of this, there have been two main difficulties; first, the failure in so many cases to complete the pathologic picture by necropsy and the microscopic study of metastases, and second, the lack of uniformity in the classification of these tumors. This makes it hard to interpret pathologic reports from different hos-

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1. Greenough, R. B.; Simmons, C. C., and Harmer, T. W.: Bone Sarcoma. Analysis of Cases from 1911 to 1921, admitted to the Massachussetts General Hospital and the Collis P. Huntington Memorial Hospital, J. Orth. Surg. **3**:602 (Nov.) 1921.

2. Morton, J. J., and Duffy, W. C.: Clinical and Pathologic Study of Ten Bone Tumors, Arch. Surg. **7**:469-531 (Nov.) 1923.

3. Meyerding, H. W.: Sarcoma of Long Bones; Study of Microscopically Proved Cases, Surg., Gynec. & Obst. **34**:321-332 (March) 1922.

4. Bloodgood, J. C.: Bone Tumors, Benign and Malignant: A Study of 370 Cases, Am. J. Surg. **34**:229-237 (Sept.) 1920; Tumors of Bone, Northwest Med. **21**:308-312 (Sept.) 1922.

5. Ewing, James: A Review and Classification of Bone Sarcomas, Arch. of Surg. **4**:485-533 (May) 1922.

pitals, as there are almost as many varieties of terminology as there are varieties of pathologists.

The first difficulty should be met by eliminating from statistical discussion any case which does not present a microscopic section of the primary tumor, and also, by impressing the profession with the importance of obtaining sections from the metastatic as well as from the primary tumor.

The solution to the second problem lies in the adoption by both clinicians and pathologists of a uniform classification. The nomenclature most commonly used in the past has been based on the histologic picture, but this has been obviously unsatisfactory. It is very difficult to make a classification that includes the elements of cellular morphology, histogenesis, anatomic origin, and clinical course, and yet all four of these factors seem inextricably involved in the differentiation of these tumors. We have used in our series a classification recently suggested by the committee on bone tumors of the American College of Surgeons. We feel that this nomenclature is simpler and possibly more practical than any generally used at the present time. We found, however, some definite disadvantages; it is by no means simple to place all of the tumors under their proper headings. We found it impossible to differentiate between periosteal, parosteal, and subperiosteal, except as indicated by the course of growth. Next, the subdivision into telangiectatic, sclerosing and undifferentiated sarcomas is obviously artificial and forces a rather arbitrary grouping. Finally, this nomenclature has the same objection which is found in the others, in that it conveys little to the surgeon regarding the clinical course of the case. This latter criticism, we realize, would probably apply to any classification, because of the uncertainty inherent in the character of the disease.

NOMENCLATURE OF BONE TUMORS

1. Metastatic tumors
2. Periosteal fibrosarcoma
3. Osteogenic tumors
    1. Benign
        a. Exostosis
        b. Osteoma
        c. Chondroma
        d. Fibroma
    2. Malignant (osteogenic sarcoma)
        A. Anatomic types
            Medullary and subperiosteal
            Periosteal
            Sclerosing
            Telangiectatic
        B. Undifferentiated sarcoma

4. Inflammatory conditions that may simulate tumors
    1. Osteoperiostitis
        a. Traumatic
        b. Syphilitic
        c. Infectious
    2. Osteitis fibrosa cystica
5. Giant cell tumor
6. Angioma
    1. Benign
    2. Malignant (angio-sarcoma)
7. Ewing's tumor (probably Endothelioma)
8. Myeloma

Our cases are confined to heading 3, subdivision 2, and headings 5 and 7.

MEDULLARY AND SUBPERIOSTEAL TUMORS

Under heading 3, subdivision 2, "Medullary and Subperiosteal," of the foregoing nomenclature fifteen cases have been tentatively grouped. Of the patients suffering from this type of tumors (Table 1) eight were males and seven, females. In age, they ranged from 15 to 40, the average being 24. One was aged 15; two, 18; one, 21; one, 23; five, 25; one, 26; one, 31; and one, 40. In five cases, the site of the tumor was the lower end of the femur; in one, the upper end of the femur; in three cases, the upper end of the tibia; in one, the upper end of the fibula; in one, the ileum; in one, the maxilla, and one, the upper end of the humerus (Fig. 1).

So many accidental factors occurred between the onset of symptoms and the admission of the patient to the hospital that the intervening time is of no statistical significance.

Seven patients gave a definite history of injury, four having a pathologic fracture. In three of these cases, the fractures antedated pain and swelling; in the other, pain and swelling antedated fracture. Six had both pain and swelling; four had swelling without pain; and three had pain without swelling.

The physical signs were without particular significance; there was usually the finding of a hard mass at the pathologic site, with or without tenderness. One gave symptoms of acute inflammation with signs of synovitis in the contiguous joint.

An analysis of clinical diagnosis in this series is not significant, as in many of the cases we do not know whether the diagnosis was made before or after the pathologic and roentgen-ray findings. The roentgen-ray diagnosis was wrong in the majority of cases in this type of tumor, the errors being osteomyelitis, destruction of bone, giant cell sarcoma and periostitis.

TABLE 1.—*Medullary and Sub-Periosteal*

| Case | Age | Sex | Admission Date | Site | Duration | Symptoms and Physical Signs | Clinical Diagnosis |
|---|---|---|---|---|---|---|---|
| 1 | 18 | M | 4/17/16 | Lower end of right femur | 6 wk. | Trauma, pain and swelling since injury; mass over lower part of right femur | Sarcoma |
| 2 | 40 | F | 11/30/16 | Sternum | 4 mo. | Swelling 4 months, no pain or tenderness; tumor mass over sternum | Sarcoma |
| 3 | 24 | M | 9/11/16 | Skull | 1 yr. | Tumor mass back of head..... | Osteoma or sarcoma |
| 4 | 17 | F | 3/13/12 | Upper end of left femur | 17 mo. | Pain outer side of left femur for 17 months; fusiform swelling upper end of femur | Sarcoma |
| 5 | 25 | M | 2/16/16 | Right ileum | 6 mo. | Strained back, pain in right hip; tumor mass attached to right ileum | Sarcoma |
| 6 | 26 | F | April, 1919 | Lower end of tibia | 3 yr. | Deep seated pain, lower part of leg 3 years; tumor mass lower end of tibia, very hard | Sarcoma of tibia and fibula |
| 7 | 17 | M | 12/ 8/15 | Upper maxilla | 1 mo. | Followed ulcerated tooth 6 months ago, no pain; swelling extending from nose to right ear | Sarcoma |
| 8 | 15 | F | 8/ 2/20 | Lower end of right femur | 3 mo. | Tumor developed 3 months following fracture; loss of function, redness, swelling and fluctuation | Sarcoma |
| 9 | 23 | M | 11/ 9/20 | Upper end of left humerus | 5 mo. | Following repeated injuries; sharp intermittent pain for past 5 months in shoulder; no swelling; atrophy of shoulder muscle, tenderness and pain on motion | Sarcoma |
| 10 | 35 | M | Oct., 1917 Aug., 1918 | Upper end of tibia | 2 yr. | Trauma; localized swelling upper end of tibia; no pain or tenderness; large semifluctuating tumor mass upper part of tibia | Giant cell sarcoma |
| 11 | 24 | M | 2/ 3/17 | Lower end of right femur | 1 yr. | Gradually increasing swelling lower end of femur, 1 year; disability and intense pain; hard fusiform swelling, lower end of right femur | Sarcoma |
| 12 | 26 | F | 4/23/16 | Lower third of right femur | 9 mo. | Following fracture, persistent pain in lower end of femur for 9 months; fracture of femur | Fracture of femur |
| 13 | 24 | F | 9/ 8/22 | Upper end of fibula | 8 mo. | Swelling in right knee; no pain; dense tumor mass, upper part of fibula | Sarcoma |
| 14 | 21 | M | 2/24/21 | Upper end of right tibia | 4 mo. | Injury, progressive swelling, pain and disability; swelling 2½ times normal, tender and edematous | Acute osteomyelitis, sarcoma |
| 15 | 31 | F | 3/ 5/16 | Upper end of tibia | 14 mo. | Swelling and pain in right knee; pathologic fracture; swelling, redness and fluctuation; enlarged lymph nodes | Sarcoma |

In the microscopic pathologic diagnosis, sections of four showed only spindle cells (Fig. 2); three showed spindle and giant cells and four showed new bone formation; three showed mixed spindle and round cells, one of which had scattered giant cells. All these tumors were very vascular, some showing large blood spaces. The presence of blood spaces in some might indicate a classification under telangiectatic, but this seems to us an artificial division. In view of the uniformly bad prognosis in this group, irrespective of histology, little can be gained from the histologic analysis, except that the tumors showing a predominence of round cells seemed to have a distinctly shorter expectancy of life.

*(Malignant Osteogenic Sarcoma)*

| Roentgen-Ray Diagnosis | Pathology, Gross and Microscopic | Pathologic Diagnosis | Treatment | Recurrences and Follow-Up |
|---|---|---|---|---|
| New bone | No gross; masses of round cells, connective tissue and bone | Osteo-sarcoma | Amputation at midthigh | Died of metastasis in lungs 8 months later |
| None | Small piece of tissue; masses of round cells with many blood spaces | Round cell sarcoma | Radium and Coley toxin | Died 1 year later, probably from general metastases |
| Newly-formed bone | Small piece of tissue; cellular mass of oval, rounded stellate cells, giant cells, calcium | Osteo-sarcoma | Exploratory operation | Died 11 months after operation, intereranial pressure |
| None | No gross; small, rounded, oval cells | Round cell sarcoma | Disarticulation at hip | Died 4 months after operation, metastases |
| Destruction of bone | Small piece of tissue; mass of small, rounded, deeply staining cells | Spindle cell sarcoma | Coley's toxin roentgen ray, radium | Died 18 months later, metastases in lungs |
| None | Large tumor mass above ankle involving tibia and fibula; mass of large spindle cells; mitotic figures | Sarcoma | 1. Curettage; 2. Amputation above knee | No recurrence until 1923; then developed large tumor mass in subclavicular region |
| None | Tissue removed for biopsy; mass of small round cells, very vascular | Round cell sarcoma | Roentgen ray and mixed toxins | Died 4 months later; general metastases |
| Destruction of bone | Joint distended with soft friable tissue; mass of spindle and cuboidal shaped cells | Mixed cell sarcoma | Amputation at midthigh | Died 8½ months after operation |
| None | Fungating growth projecting from anatomic neck; fracture; mass of spindle cells, occasional giant cell, and bone; muscle infiltrated | Spindle cell sarcoma | Interscapular thoracic amputation | Died 21 months after operation; metastasis in chest; necropsy |
| None | Large lobulated tumor mass involving medullary cavity and invading soft parts; mass of spindle and oval shaped cells | Mixed, spindle and round cell sarcoma | 1. Curettage; 2. Amputation above knee | Died 36 months after curettage; abdominal metastasis |
| ..... | Tumor mass invading medullary cavity; masses of spindle cells | Periosteal sarcoma | Roentgen ray and radium curettage | Progressive metastasis in lung |
| Pathologic fracture, giant cell sarcoma | Curettings and nodule in arm; connective tissue, bone, cellular areas, scattered giant cells; specimens were vascular | Mixed cell sarcoma | Curettage | General metastasis; died 8 months after admission |
| Osteomyelitis | Leg at midthigh; firm tumor mass at head of femur involving medullary cavity and soft parts; small spindle and polyhedral cells | Periosteal sarcoma | Amputation | Four months after admission large tumor mass occupying lung and mediastinum |
| Destructive osteoarthritis | Tissue removed for biopsy; spindle and round cells | Spindle and round cell sarcoma | None | Progressive; died 1 month after admission; necropsy, general metastasis |
| Sarcoma | Tumor mass lower end of femur involving medullary cavity and joint; masses of spindle cells | Spindle cell sarcoma | Amputation at midthigh | Died 9 months after admission; metastasis in lungs |

Eight patients were treated by amputation; four, by roentgen-ray, three of them having, in addition, Coley's serum and two, radium; one was curetted and two had simple explorations. In view of the one hundred per cent. mortality or recurrence, no advantage can be placed on any type of treatment.

All these patients but one, died of metastases within a period ranging from two to thirty-nine months after their original admission to the hospital for treatment. It is not possible to find the exact condition at death without necropsy; but as a rule, metastases are in the lung. The one outstanding feature of this group of cases is the uniformly fatal prognosis.

PERIOSTEAL TUMORS

These two cases (Table 2) have been placed under this heading, as the tumors grew from the periosteum into the soft parts, rather than from the periosteum into the bone (Figs. 3, 4). Both had a rapidly

Fig. 1 (Case 9).—Medullary sarcoma of upper end of humerus. Death by pulmonary metastases.

progressive course in spite of amputation, with death from pulmonary and general metastases. The histology did not coincide in the two cases; one growth was made up of bone, cartilage and masses of round and spindle cells with many blood spaces (Fig. 5); the other consisted entirely of compact masses of spindle cells.

THE SCLEROSING TYPE OF TUMORS

In grouping cases (Table 3) under this heading, we may have misinterpreted the instructions of the Committee. We have selected those cases which, microscopically, showed a comparatively adult type of

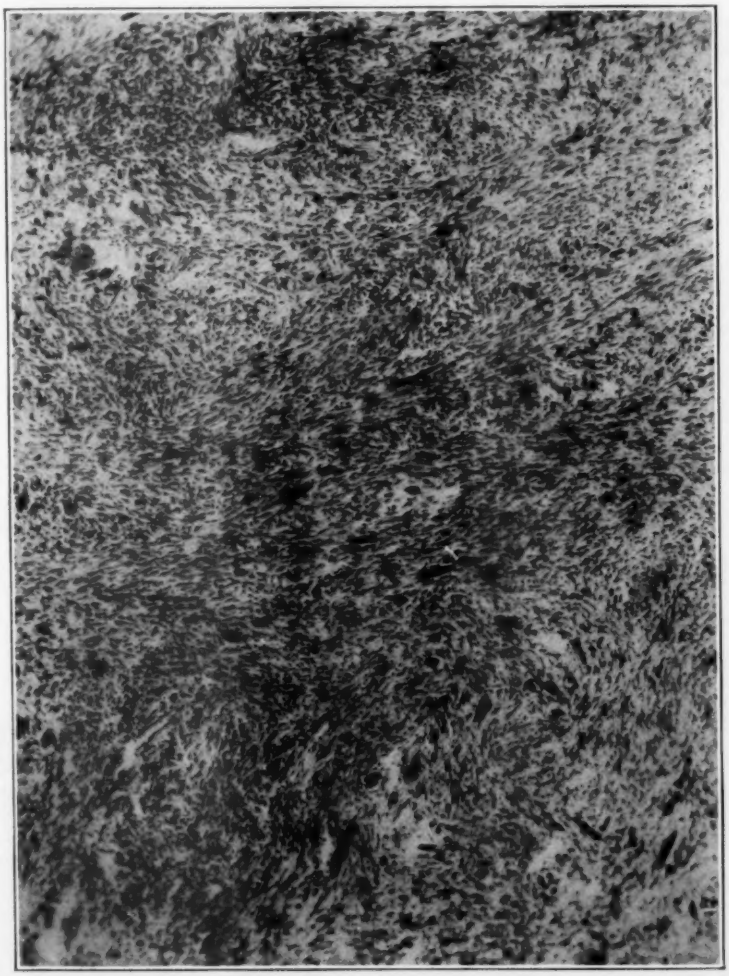

Fig. 2 (Case 9).—Photomicrograph of section from specimen shown in Figure 1.

tissue, and have not been influenced by gross clinical features. The distinction between these and medullary tumors is quite arbitrary. We included six cases, among them, tumors formerly called chondrosarcoma and fibrosarcoma.

TABLE 2.—

| Case | Age | Sex | Admission Date | Site | Duration | Symptoms and Physical Signs | Clinical Diagnosis |
|---|---|---|---|---|---|---|---|
| 16 | 26 | M | 7/15/16 | Shaft of humerus | 2 mo. | Intense pain and swelling over shaft of humerus; large dense tumor mass, upper third of humerus | Sarcoma |
| 17 | 35 | F | 5/11/20 | Lower end of right femur | 2 mo. | Pain and swelling in right knee; tumor mass, lower end of right femur; very dense | Sarcoma |

TABLE 3.—

| Case | Age | Sex | Admission Date | Site | Duration | Symptoms and Physical Signs | Clinical Diagnosis |
|---|---|---|---|---|---|---|---|
| 18 | 28 | F | 10/27/16 | Spine of scapula | 7 mo. | Pain and stiffness in shoulder; tumor mass attached to spine of scapula | Sarcoma |
| 19 | 46 | F | 11/18/15 | Middle shaft of femur, right | 2 yr. | Pain and swelling; swelling middle shaft of right femur | ..... |
| 20 | 27 | F | 11/19/21 | Upper maxilla, right | 2½ mo. | Following extraction of tooth, rapidly increasing swelling in upper jaw; pain severe; firm tumor mass 6 cm. in diameter attached to upper jaw | Giant cell tumor |
| 21 | 37 | M | 6/11/17<br>10/18/17<br>11/ 6/19 | Upper end of tibia | 2 mo. | Pain and swelling; tumor mass, upper third of tibia, tender and fluctuating | Sarcoma |
| 22 | 27 | M | 1/15/19<br>10/ 2/19<br>2/16/20 | Upper end of humerus | 1½ yr. | Pain and loss of function; shoulder swollen, tender and warm | Sarcoma |
| 23 | 13 | M | 2/14/20 | Shaft of left humerus | 1 wk. | Following injury, pathologic fracture; no tumor mass felt | Sarcoma |

TABLE 4.—

| Case | Age | Sex | Admission Date | Site | Duration | Symptoms and Physical Signs | Clinical Diagnosis |
|---|---|---|---|---|---|---|---|
| 24 | 24 | M | 4/15/18 | Ileum | 5 mo. | Severe pain over ileum; hard round tender mass attached to right iliac crest | Sarcoma |

TABLE 5.—

| Case | Age | Sex | Admission Date | Site | Duration | Symptoms and Physical Signs | Clinical Diagnosis |
|---|---|---|---|---|---|---|---|
| 25 | 27 | M | 5/31/21 | Lower fourth of femur | 4 mo. | Swelling and pain in left knee; marked swelling around inner aspect of joint; slightly tender | Sarcoma of femur |
| 26 | 52 | F | 9/ 7/21 | Lower end of right radius | 4 mo. | Gradual swelling, lower end of radius; rheumatic pains in wrist; hard bony swelling encircling right wrist, 11 cm. in diameter; no pain on motion | Myeloma |
| 27 | 25 | F | 4/26/20<br>3/29/21 | Upper part of left tibia | 3 yr. | On 4/26/20, operated on for osteomyelitis of tibia; 3/29/21, pain and tumor mass in old scar; tumor mass just below tuberosity of tibia | Sarcoma of tibia |

*Periosteal*

| Roentgen-Ray Diagnosis | Pathology, Gross and Microscopic | Pathologic Diagnosis | Treatment | Recurrences and Follow-Up |
|---|---|---|---|---|
| None | Large tumor mass encircling upper third of humerus, attached to periosteum but not invading bone; large rounded and spindle cells, bone and cartilage; very vascular | Osteosarcoma | Disarticulation of arm and shoulder blade | Died 3 months after admission; metastasis in lungs |
| Periosteal sarcoma | Large dense tumor mass occupying lower end of femur; masses of spindle cells | Spindle cell sarcoma | Amputation at hip | Died 18 months after admission; metastasis in lungs |

*Sclerosing*

| Roentgen-Ray Diagnosis | Pathology, Gross and Microscopic | Pathologic Diagnosis | Treatment | Recurrences and Follow-Up |
|---|---|---|---|---|
| None | Firm, light colored tumor mass attached to spine of scapula; mass of elongated cells; vascular | Spindle cell sarcoma | Excision of tumor mass with part of spine of scapula | Recurred almost immediately; again excised; no recurrence to April, 1922 |
| Sarcoma | Encapsulated subperiosteal tumor; spindle cells and a few scattered giant cells | 1. Spindle cell sarcoma; 2. giant cell sarcoma | Excision of encapsulated mass | No recurrence after 7 years |
| Tumor of jaw | Two encapsulated, lobulated tumor-like masses of firm consistency; richly cellular mass of connective tissue; picture suggests richly cellular fibromyoma of uterus | Fibrosarcoma | Excision and radium | Recurred almost immediately; died 6 months after admission; extensive local recurrence |
| Tumor of tibia | Tumor mass, upper third of tibia; soft fibrous tissue; giant cells | Fibrosarcoma | Curettage; amputation at midthigh | Metastasis glands in inguinal region; died 2½ years after first admission |
| Fracture of humerus | Tumor mass upper third of humerus invading medullary cavity; cellular connective tissue, bone and cartilage | Osteochondrosarcoma | Resection of upper third of humerus | No recurrence after 4 years |
| Rarefaction of bone | Curettings; spindle or oat-shaped cells with collagen fibers; vascular | Fibrosarcoma | Curettage | No recurrence, 1922 |

*Telangiectatic*

| Roentgen-Ray Diagnosis | Pathology, Gross and Microscopic | Pathologic Diagnosis | Treatment | Recurrences and Follow-Up |
|---|---|---|---|---|
| Sarcoma | Tissue for biopsy; large round cells; very vascular | Round cell sarcoma | ..... | Died 5 months after admission; metastasis |

*Undifferentiated*

| Roentgen-Ray Diagnosis | Pathology, Gross and Microscopic | Pathologic Diagnosis | Treatment | Recurrences and Follow-Up |
|---|---|---|---|---|
| Giant cell sarcoma | (1) tissue for biopsy; (2) leg at midthigh; large tumor mass extending from lower fourth of femur to cartilage, invading medullary cavity and surrounding tissues; bone cavity showed small giant cells, round cells, spindle cells; soft parts showed spindle cells and bone | Sarcoma | Curettage and amputation at midthigh | No recurrence 2½ years after operation |
| Medullary sarcoma | Large tumor mass occupying lower extremity of radius, invading medullary cavity and infiltrating soft parts; bone cavity showed spindle cells, ovoid cells and blood vessels; soft parts showed spindle cells and bone | Sarcoma | Amputation upper third of forearm | No recurrence 2½ years after operation |
| Sarcoma | Large and small spindle and round cells | Mixed cell sarcoma | Curettage and amputation | Recurrence 11 months after curettage; January, 1923, no recurrences; patient had gained in weight, and wore an artificial leg |

Fig. 3 (Case 16).—Periosteal tumor of right arm before amputation.

Fig. 4 (Case 16).—Vertical section of humerus after amputation showing growth of periosteal tumor into soft parts, rather than into shaft.

The average age of the patients in this group was 35 years, considerably higher than that of the former group. Only two patients died, one of metastases of the lymph nodes three years after admission, and one of local recurrence. The last was a case (Case 20) in which a well

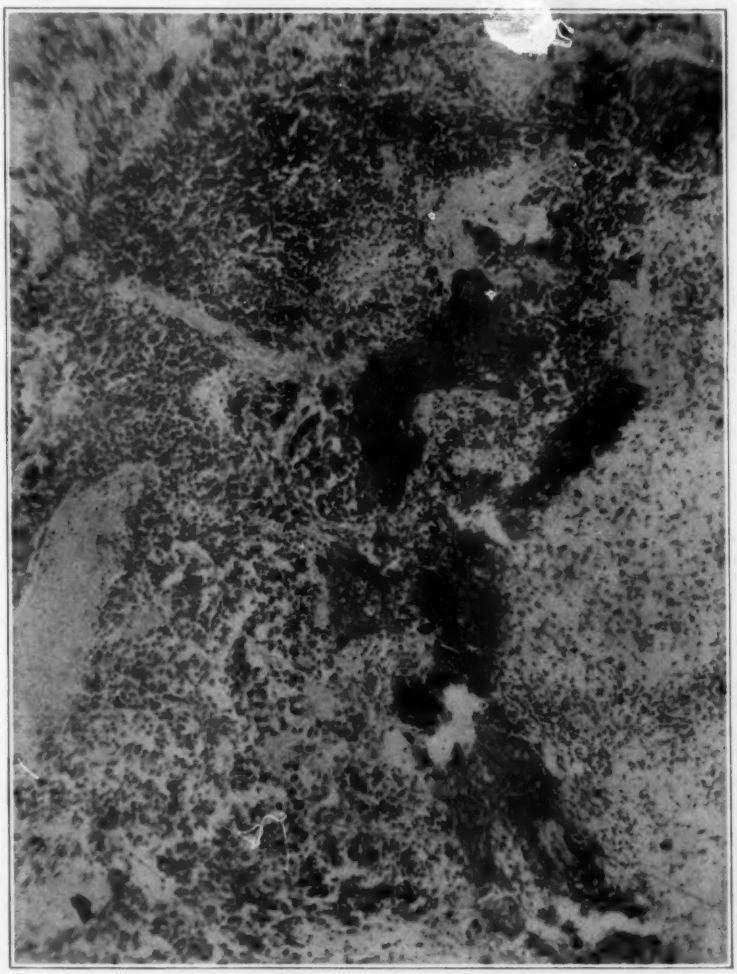

Fig. 5 (Case 16).—Masses of round and spindle cells, with new bone and cartilage formation, in periosteal tumor.

encapsulated fibrosarcoma was removed from the antrum of Highmore apparently intact, only to recur locally in a few weeks and invade the orbit and base of the brain. One of the patients who died was treated by amputation; three, now alive and well, were treated by excision. In one case, there was a recurrence successfully excised, and one case

was treated by resection of the humerus and a bone transplant. There is a distinctly hopeful prognosis to the cases in this division (Figs. 6, 7, 8, 9, 10).

Fig. 6 (Case 18).—Spindle cell sarcoma, sclerosing type, from spine of scapula. Note invasion of muscle. Excision in 1916. No recurrence.

Greenough, Simmons and Harmer,[1] in a recent group analysis, reported three cases of osteogenic sarcoma in which the patients were alive after five years. The histology as published would indicate that in this present classification they would probably fall in this sclerosing group.

THE TELANGIECTATIC TYPE

Under the telangiectatic type we have arbitrarily placed one case (Case 24, Table 4) which corresponds to what we would formerly have called a vascular round cell sarcoma with a very rapid fatal termination. The main point of interest in this case is the resemblance between the cells in this tumor and those in the so-called endothelioma or Ewing's

Fig. 7 (Case 19).—Encapsulated, subperiosteal tumor, sclerosing type. No recurrence seven years after excision.

tumor. Judging from the slide alone (Fig. 11), it would be very hard to keep this tumor out of the latter classification.

THE UNDIFFERENTIATED TYPE

These three cases were grouped as belonging to the undifferentiated type, because sections showed a mass of undifferentiated cells with no single type predominating. It is hard to draw a conclusion from only three cases, but apparently this is a more benign type of tumor of this

type, as all three patients are alive and well, two having been living about three years (Figs.12, 14), and one about five years since treatment. All three growths had broken through their capsules and into the soft parts (Fig. 15). In two, there was new bone formation in the midst of masses

Fig. 8 (Case 22).—Medullary sarcoma of upper end of humerus, sclerosing type. No recurrence four and one-half years after excision.

of small spindle cells. One showed a few small giant cells in the bone cavity, but lacked the other characteristics of a giant cell tumor. In two of these cases (Cases 26 and 27, Table 5), a prognosis based on a study of the section would have been absolutely misleading, as they both presented the microscopic features (Figs. 13, 16) of extreme malignancy.

The formation of new bone in the tumor mass has been associated with malignancy, but this feature seemed of no great significance in these cases.

If we consider all the preceding cases without subdivision, but under the main heading "Malignant Osteogenic Sarcoma," we find that of

Fig. 9 (Case 22).—New bone formation and spindle cells in rather adult stroma, from a section from the specimen shown in Fig. 8.

twenty-seven patients, seven are alive and free from recurrence. One has passed the eight year period; one, seven years; three, four years; one, three years, and one, two and one-half years. This is an unexpectedly low mortality and this fact is discussed later under general prognosis.

GIANT CELL TUMOR

This forms the largest single group (Table 6) of our series, although we have included none of those tumors known as epulis of the jaw. We have confined ourselves in this group to the tumors of the long

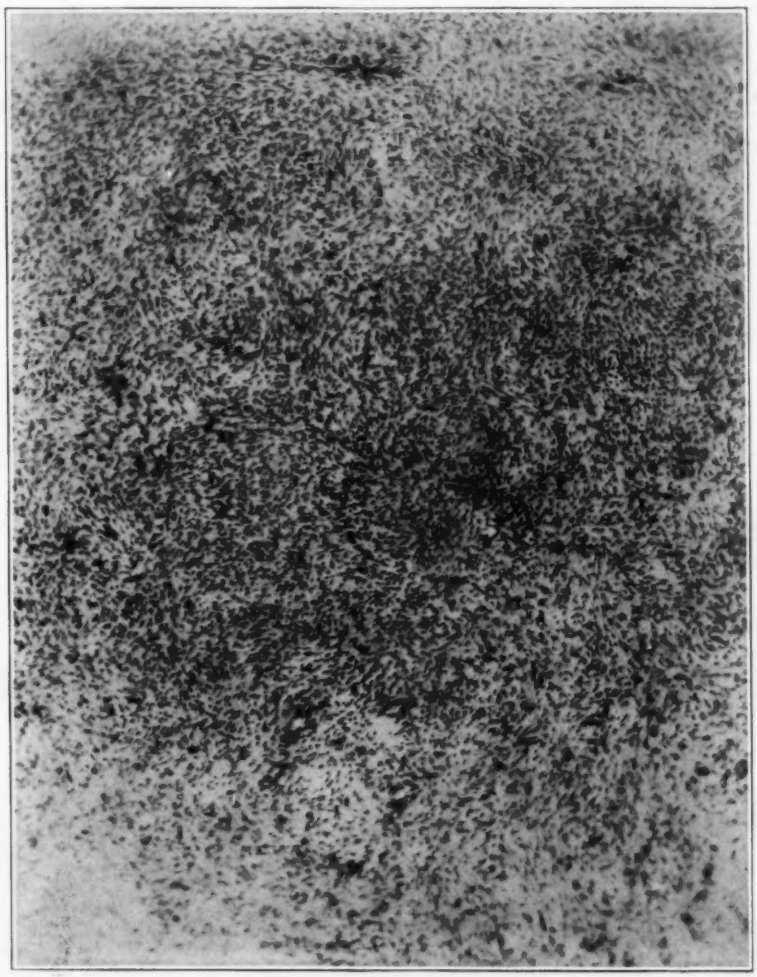

Fig. 10 (Case 23).—Sarcoma of shaft of humerus (sclerosing type?). Although it was fairly cellular, the patient shows no evidence of recurrence four years after curettage.

bones. The majority of these could be classified without difficulty, but there remained a considerable minority whose inclusion in this group might be open to question. This very difficulty of classification we considered of greatest importance.

The group is first analyzed as a whole. The age varies from 10 to 50 years, the average being 27 years. Twelve patients were females, and eight, males. There were the usual symptoms of pain and swelling; three suffered from a pathologic fracture; ten gave a definite history of

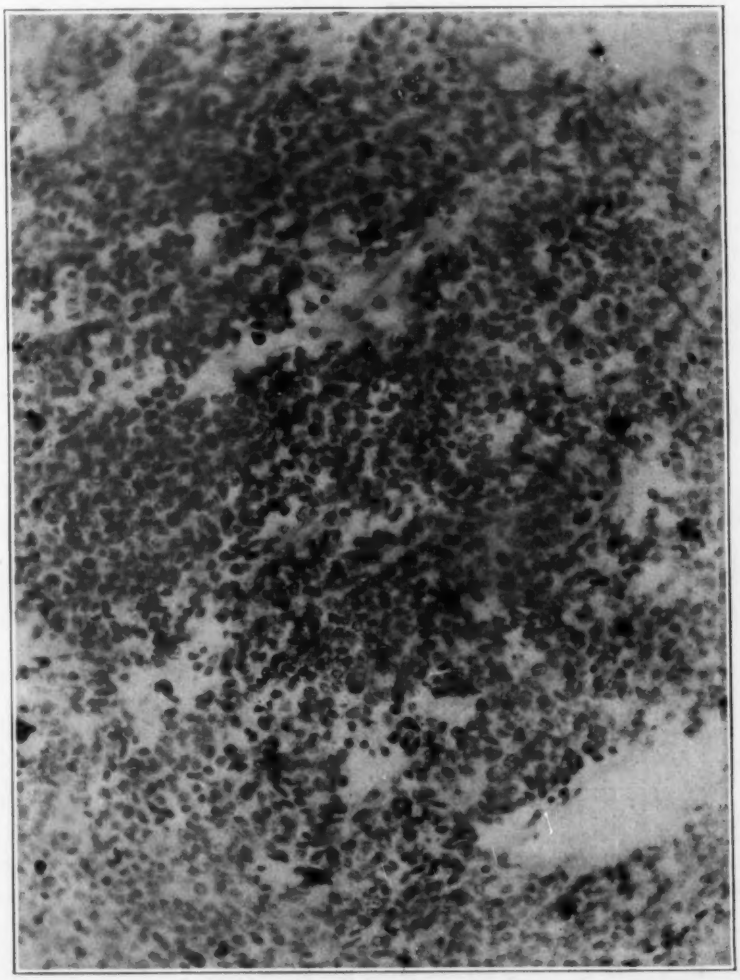

Fig. 11 (Case 24).—Section from telangiectatic sarcoma of ilium and internal shaft of long bone. Round cells grouped around blood vessels, as in Ewing's tumor. Very malignant.

injury. In the clinical and roentgen-ray diagnoses there were a large number of errors, and, in some cases, there was a diagnosis of sarcoma without differentiation. The most common error was in the diagnosis of osteomyelitis. The pathologic diagnosis in fifteen cases was giant

TABLE 6.—*Giant*

| Case | Age | Sex | Admission Date | Site | Duration | Symptoms and Physical Signs | Clinical Diagnosis |
|---|---|---|---|---|---|---|---|
| 28 | 44 | F | 4/ 8/14 | Lower end of left femur | 1 yr. | Pain and disability of left knee; left knee swollen and red | ..... |
| 29 | 18 | F | 2/ 2/16 | Lower end of femur | 6 yr. | Fracture of leg 6 years ago; 1 year later tumor developed at site; 1 month before admission, pain and cough in left chest | Metastases in chest |
| 30 | 18 | F | 8/13/20 | Middle third left tibia | 6 mo. | Tumor mass middle third of left tibia | Sarcoma of tibia |
| 31 | 29 | M | 10/28/21 | Lower end of femur | 20 mo. | Pain and swelling ..... | ..... |
| 32 | 28 | F | 7/19/20 | Lower end of femur | 2 yr. | Pain in left leg and knee; tender swelling around knee, loss of function | Osteo-sarcoma |
| 33 | 11 | F | 6/15/15 | Middle finger | 3 or 4 yr. | No pain or loss of function; hard elastic growth, inner side of right middle finger | Sarcoma of finger |
| 34 | 50 | F | Oct., 1921 | Head of left tibia | 2 yr. | Sharp pain around knee; large excavated area head of tibia, filled with a tumor mass which protrudes beyond skin | Sarcoma |
| 35 | 42 | F | 12/ 1/19 | Upper third of tibia | 8 yr. | Injury to knee 12 years ago; 4 years later, painful swelling inner side of knee; for the past 4 years this has increased in size; large mass over upper end of tibia, firm, hot and fluctuating | Giant cell sarcoma of tibia |
| 36 | 35 | M | 6/14/22 | Head of tibia | 18 mo. | Swelling and rheumatic pains in knee; hard circumscribed tumor mass over external tuberosity of tibia; no tenderness | Giant cell sarcoma |
| 37 | 18 | M | 1/ 3/16 | Lower end of femur | 5 mo. | Following injury, developed large mass in lower third of femur; large tumor, lower third of femur | Sarcoma of femur |
| 38 | 10 | F | 3/28/16<br>4/22/16<br>10/13/16 | Uppen end of tibia | 3 wk. | Pain and swelling following injury; slight swelling upper end of tibia; tender | Osteo-myelitis |
| 39 | 27 | M | 2/ 4/15 | Upper end of tibia, left | 8 yr. | Following trauma, swelling and synovitis of knee; tender swelling, upper part of tibia | Myeloma |
| 40 | 26 | M | 5/18/16 | Lower end of femur | 3 yr. | Following injury, swelling and pain in knee; tumor mass lower end of femur | Sarcoma |
| 41 | 18 | F | 2/15/20 | Upper end of ulna | 1 mo. | Following injury, pain, deformity, loss of function; swelling over upper end of ulna | Bone cyst |
| 42 | 46 | F | 3/ 4/20 | Lower end of right radius | 4 mo. | Pain, swelling and tenderness..... | Sarcoma |
| 43 | 21 | M | 4/21/19 | Lower end of right ulna | 1 mo. | Swelling and pain; tumor mass lower end of right ulna; loss of function | Sarcoma |
| 44 | 28 | F | 5/28/17<br>4/ 2/18 | Lower end of radius | 2 mo. | Pain following injury; swelling, redness and tenderness around wrist joint | Cyst of radius |
| 45 | 23 | F | 3/31/18 | Upper end of left tibia | 2 mo. | Pain, redness, tenderness and fluctuation | Osteo-myelitis |
| 46 | 27 | M | 2/22/19 | Lower end of right femur | 5 yr. | Swelling and pathologic fracture..... | Giant cell sarcoma |
| 47 | 38 | F | 2/25/18 | Shaft of femur | 4 mo. | Swelling and weakness; tumor mass, lower end of femur; pathologic fracture | Giant cell sarcoma; tuberculosis; gumma |

| Roentgen-Ray Diagnosis | Pathology, Gross and Microscopic | Pathologic Diagnosis | Treatment | Recurrences and Follow-Up |
|---|---|---|---|---|
| Osteosarcoma or bone cyst Metastases in chest | Curettage; connective tissue round cells and giant cells<br>Diffuse tumor in left lung; circumscribed nodules in right lung: left lung, diffuse spindle cell sarcoma; right lung, large multinucleated giant cells, spindle cells | Myeloma of femur<br>Spindle and giant cell sarcoma | Curettage and carbolization<br>Amputation of leg 5 years ago | Died of pneumonia, May 11, 1914<br>Died 6 years after curettage; metastatses in both lungs |
| Extensive myeloma of bone cyst | Curettings; large spindle cells and giant cells | Giant cell sarcoma | Curettage | No recurrence after 3 years |
| Medullary sarcoma | Right lower extremity; tumor mass lower third shaft of femur; elongated spindle and giant cells | Giant cell sarcoma | Amputation of leg | No recurrence after 2 years |
| Sarcoma | Leg at midthigh; large tumor mass, involving medullary cavity and joint; large interlacing spindle cells and giant cells | Giant cell sarcoma | Amputation | No recurrence after 3 years |
| ..... | Lobulated firm mass attached to middle finger; large spindle cells, small giant cells | Giant cell sarcoma | Excision of tumor | No recurrence after 21 months |
| Extensive bone destruction, head of left tibia; rarefaction of patella and femur | Curettage; amputation at midthigh; tumor mass, upper end of tibia, involving medullary cavity; secondary tumor involving stump; sections from primary growth, secondary growth and stump show spindle cells and multinucleated giant cells | Giant cell sarcoma | Curettage; amputation at midthigh 8 months later; radium and roentgen ray | Tumor recurred after curettage; later recurred in stump at midthigh; died 21 months after first curettage, following extensive recurrence involving thigh and pelvis |
| Giant cell sarcoma | (1) curettage; (2) amputation of leg above knee; large tumor mass head of tibia, invading soft parts; (1) showed giant cells and spindle cells; (2) giant cells and spindle cells; soft parts: spindle cells and bone | (1) Giant cell sarcoma; (2) mixed cell sarcoma | Curettage, carbolization and amputation | Immediate recurrence after curettage; no recurrence after 3 years and 9 months following amputation |
| Diagnosis of tumor not made | Mass of curettings; large giant cells and spindle cells | Giant cell sarcoma, epulis type | Curettage; amputation for septic arthritis | No recurrence 1½ years after curettage |
| Sarcoma | Lower end of femur showed soft tumor mass destroying medullary cavity and joint; spindle cells, round and oval cells; small giant cells | Giant cell sarcoma | Curettage, followed by amputation 3 months later | Recurred following curettage; recurrence in inguinal glands and large mass in abdomen; died |
| Osteomyelitis | Curettage; amputation at midthigh, medullary tumor mass, upper end of tibia; mixed cells of epulis type | Sarcoma | Curettage; amputation | Recurred following curettage; died 7 months after admission; metastasis in lungs; no necropsy |
| Disease of upper end of tibia | Mass of curettings; giant cell sarcoma | Giant cell sarcoma | Curettage, drainage; no cauterization | No recurrence after 8 years |
| Sarcoma | Curettings; giant cell sarcoma | Giant cell sarcoma | Curettage; cauterization | No recurrence after 3 years and 4 months |
| Bone cyst | Curettings; epulis type giant cells; spindle and endothelial cells | Bone cyst | Curettage | No recurrence so far as known to date |
| Sarcoma | Fusiform swelling involving lower end of radius, not involving joint; giant cell sarcoma of epulis type | Giant cell sarcoma | Resection of radius | Local recurrence after 8 months in tendon sheath; following excision and cauterization, no recurrence to date |
| None | Tumor mass upper end of ulna; giant cell sarcoma | Giant cell sarcoma | Excision head of radius | No recurrence after 3 years |
| Tuberculosis of wrist | Curettage; large multinucleated giant and a mass of large interlacing spindle cells | Giant cell sarcoma | Curettage | Recurred after 2 months; again curetted for recurrence; no recurrence to date |
| Osteomyelitis | Curettings; spindle and giant cells of epulis type | Giant cell sarcoma | Curettage and carbolization | No recurrence after 5½ years |
| Pathologic fracture; bone cyst | Amputation at midthigh; tumor mass lower end of femur; spindle cells, giant cells and new-formed bone | Giant cell tumor | Amputation at midthigh | No recurrence to date |
| Medullary sarcoma | Amputation at midthigh; tumor mass, lower end of tibia, destroying entire cortex; cellular connective tissue and multinucleated giant cells | Giant cell sarcoma | Amputation at midthigh | No recurrence to date |

cell sarcoma; one was diagnosed as a case of osteosarcoma; one, as a case of myeloma; one, as central osteogenetic sarcoma; one, as a bone cyst, and one tumor was simply called a sarcoma.

In five patients, amputation without preliminary curettage was done; in six patients curettage and cauterization were employed, without sub-

Fig. 12 (Case 25).—Undifferentiated sarcoma of lower end of shaft of femur. No evidence of recurrence three years after amputation.

sequent amputation. On six, amputation was performed at different periods subsequent to recurrence after curettage. In three cases resection was used. Of those patients curetted without subsequent amputation, one developed pneumonia following a pathologic fracture which occurred on attempting to walk too soon after curettage. The other five

are alive and well, after a period of from two to five years following the operation. One of those on whom amputation was done is free from recurrence, two years after the operation; one is free from recurrence three years after the amputation, one four years after, and one five years after. One died five years later of metastases in the chest. Of

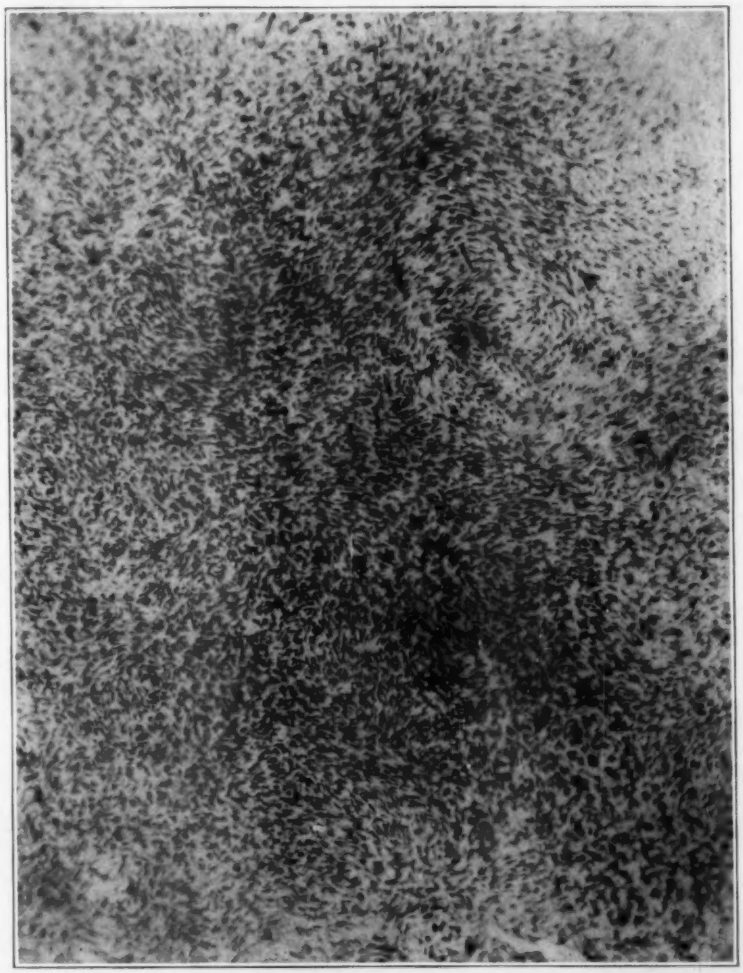

Fig 13 (Case 25).—Tumor shown in Figure 12; note the great cellularity and the absence of giant cells. Clinically, it resembled a giant cell tumor, but microscopically it was utterly dissimilar.

those patients in whose cases curettage and subsequent amputation for recurrence was employed, one is alive and well, eighteen months after the amputation, the amputation having been done for a septic knee joint following curettage; in the case of two amputations for local recurrence

Fig. 14 (Case 26).—Undifferentiated sarcoma of radius. No recurrence two and one-half years after amputation.

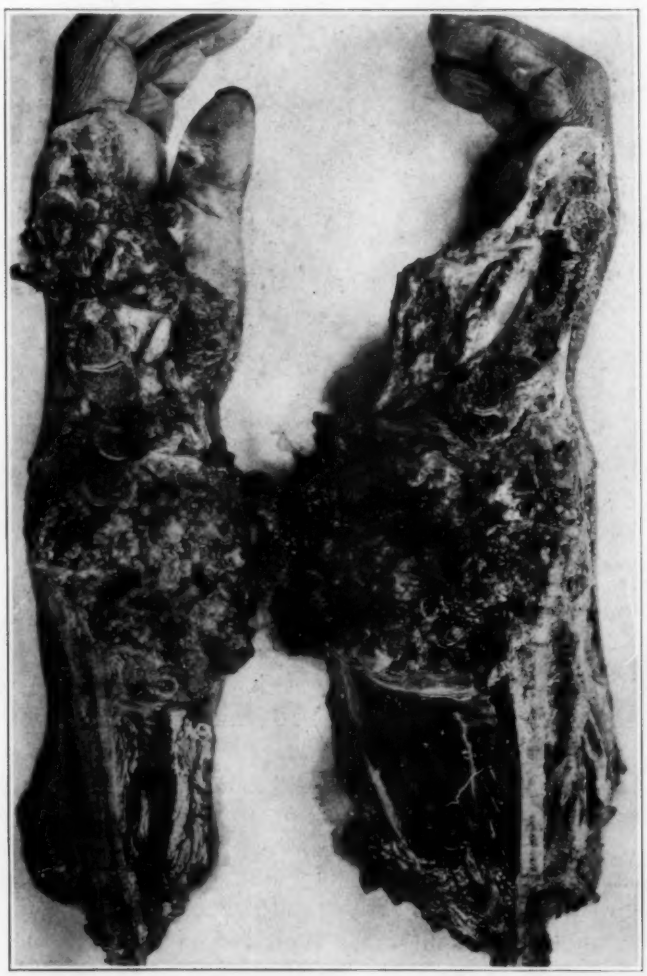

Fig. 15 (Case 26).—Vertical section of forearm shown in Figure 14. Destruction of shaft of radius with extensive invasion of the soft parts.

after curettage, both patients are free from recurrence, one after a period of three and one-half years, and the other after almost three years. One patient died twenty-one months after the first operation with an extensive recurrence in the stump at the midthigh, the original tumor having been in the tibia. One died of metastases occurring

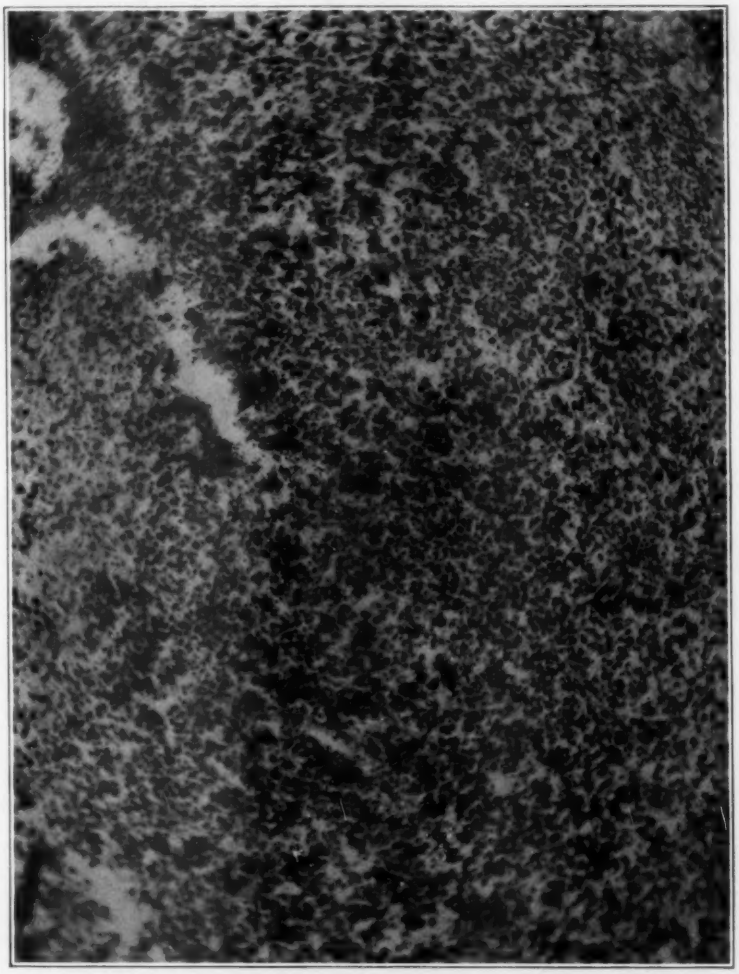

Fig. 16 (Case 26).—Section from specimen shown in Figure 15. This is a very cellular growth with predominence of no particular cell.

shortly after amputation had been performed for recurrence. One died five years after amputation from pulmonary metastases. No section of the original tumor in this latter case was obtained, as the amputation was done at another hospital, and the necropsy performed at the Presbyterian Hospital.

The able and painstaking investigations of Dr. Bloodgood in the last twenty-odd years have clarified to some extent the confusion existing in regard to this very unusual type of tumor; but we feel that there are certain characteristics as exemplified in several of our cases which have not yet been defined. The majority of these tumors follow certain

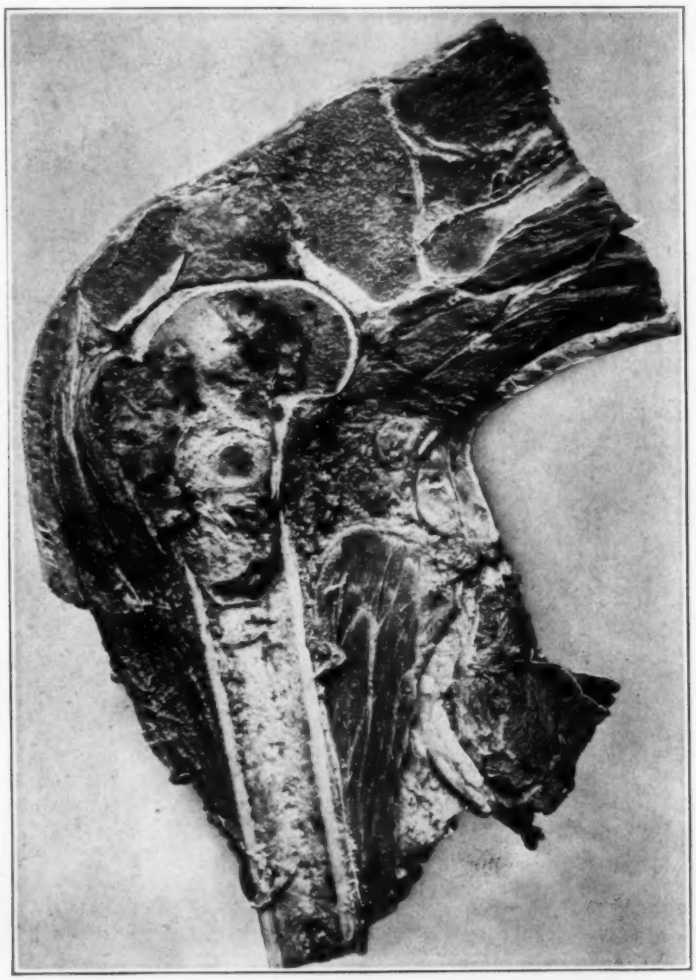

Fig. 17 (Case 31.—Giant cell tumor of lower end of femur, benign type.

definite rules. They have a characteristic clinical course and roentgen-ray picture; they rarely invade the soft parts (Fig. 17), do not metastasize, and have a rather typical histologic appearance (Fig. 18), the outstanding feature of which is the presence of large multinucleated giant cells (Fig. 20) embedded in either dense or loosely woven fibrous

stroma of comparatively adult type. This is the type that responds to curettage (Fig. 19) and is benign in character. On the other hand, there is a group which may or may not have similar clinical and roentgen-ray findings and physical signs, but in which the histologic picture is atypical. From the study of our cases, we would say that the malignancy of this

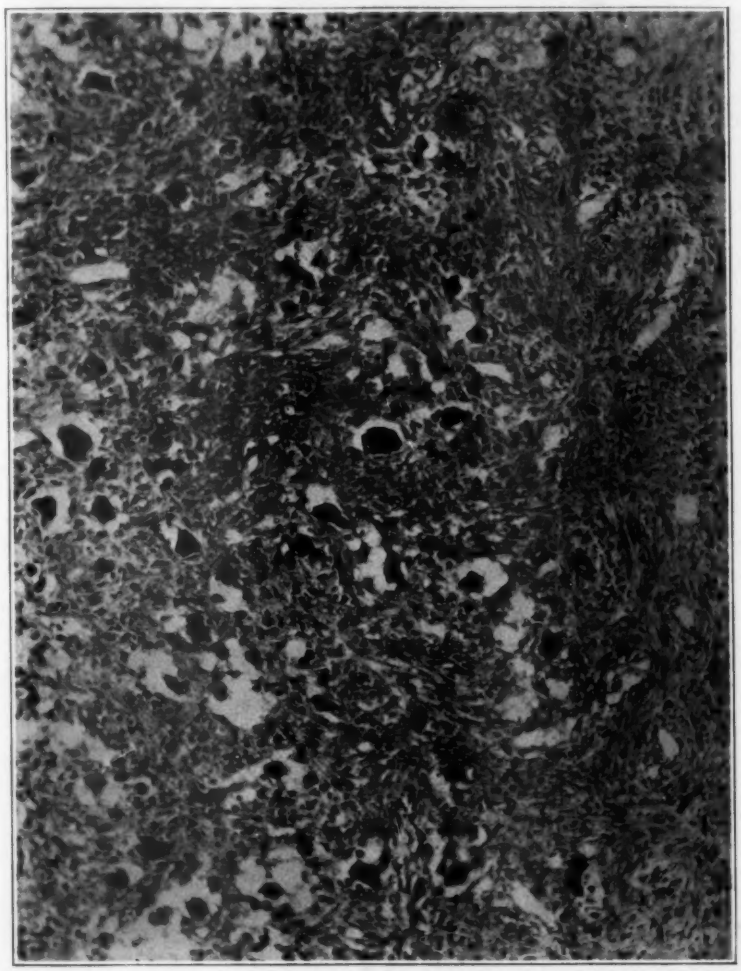

Fig. 18 (Case 31).—Section from tumor shown in Figure 17 showing masses of giant cells in a loosely woven stroma.

type, in general, varies in proportion to the cellularity of the histologic picture. There is in this latter type, besides cellularity, an increase in mitosis. In some cases, there is a marked decrease in the number and size of the giant cells. Both types show definite exceptions to this description. Case 34 is the most unusual case of the whole series. This

patient, a woman aged 50, with an eighteen months' history of pain and swelling at the upper end of the tibia, was curetted on a diagnosis of giant cell sarcoma. The roentgen-ray and histologic picture were interpreted as confirming the diagnosis. After a local recurrence (Figs. 22,

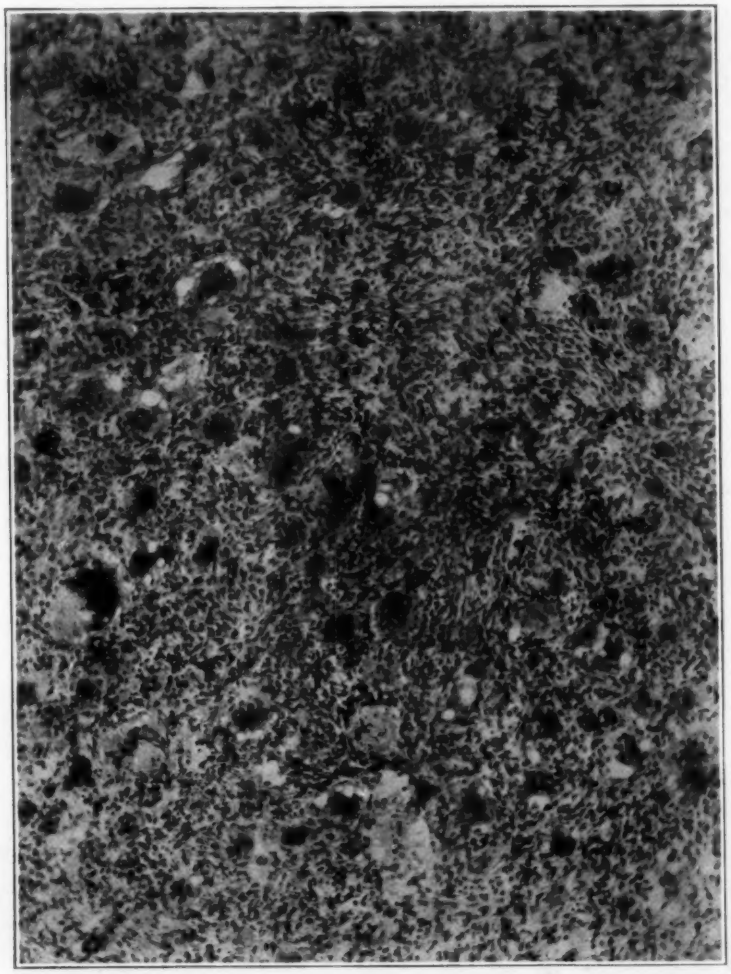

Fig. 19 (Case 40).—A very typical picture of a giant cell tumor of the lower end of femur. The patient was cured by curettage.

23), she was curetted again, radium was applied to the cavity, and systematic roentgen-ray therapy instituted. In spite of this, a recurrence again developed, and the limb was amputated (Fig. 24) at the midthigh. Shortly afterwards, there was an extensive involvement of the stump (Fig. 25) and the soft parts with tumor tissue which, on section

(Fig. 27), showed an increase in cellularity and a diminished number of giant cells, but a persistent similarity to the primary tumor (Fig. 26). This grew rapidly and she died twenty-one months after the first operation with extension to the pelvis. As no necropsy was obtained, we are

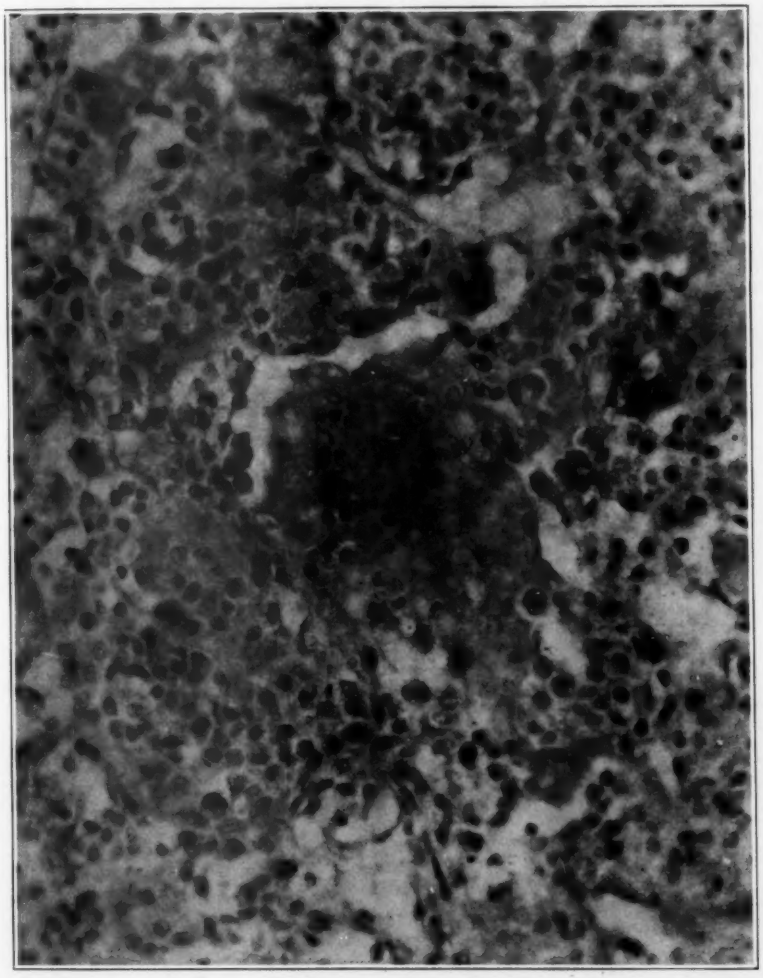

Fig. 20 (Case 47).—High power of a typical giant cell from benign giant cell tumor from the lower end of the femur.

ignorant of the presence or absence of distal metastases; but the roentgen-ray examination of the lungs and bones was negative.

There have been other instances of giant cell tumors with rapid recurrence after curettage, and these recurrences always showed marked histologic changes, but this is the only case that we know of in which

an amputation stump was involved with a recurrence of giant cell tumor showing a rapidly increasing malignancy and extensive soft part involvement. Histologically, the recurrence showed definite increase in cellularity, but many giant cells containing from eight to ten nuclei were still

Fig. 21 (Case 29).—Section from pulmonary metastases in right lung occurring five years after amputation of a supposed giant cell tumor.

present. The cells in the stroma were of both the spindle and the round cell type. Should this be called a recurrence or a metastasis?

There is one other case which could plausibly be put in a group with the preceding case, but unfortunately, a section of the primary tumor is missing. This patient, a girl aged 18, six years before admission, had

Fig. 22 (Case 34).—Recurrence in a giant cell tumor from head of tibia.

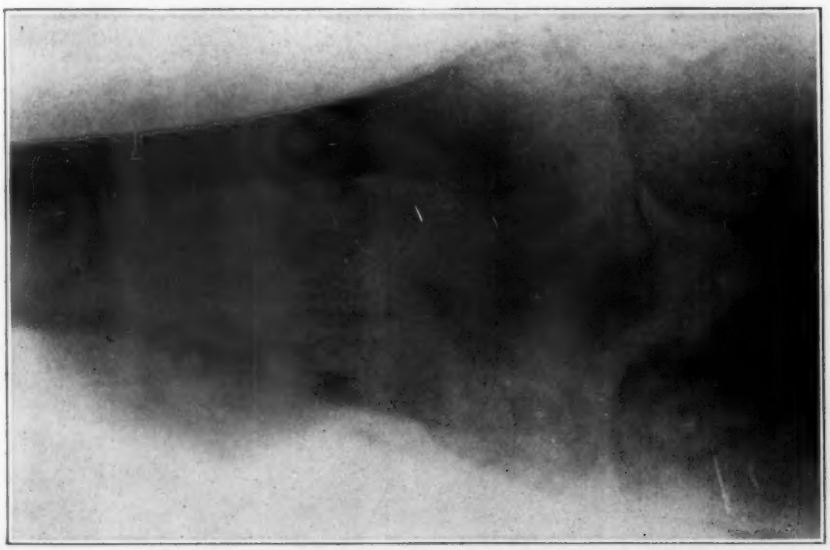

Fig. 23 (Case 34).—X-ray picture of recurrence in the head of the tibia of giant cell tumor shown in Figure 22.

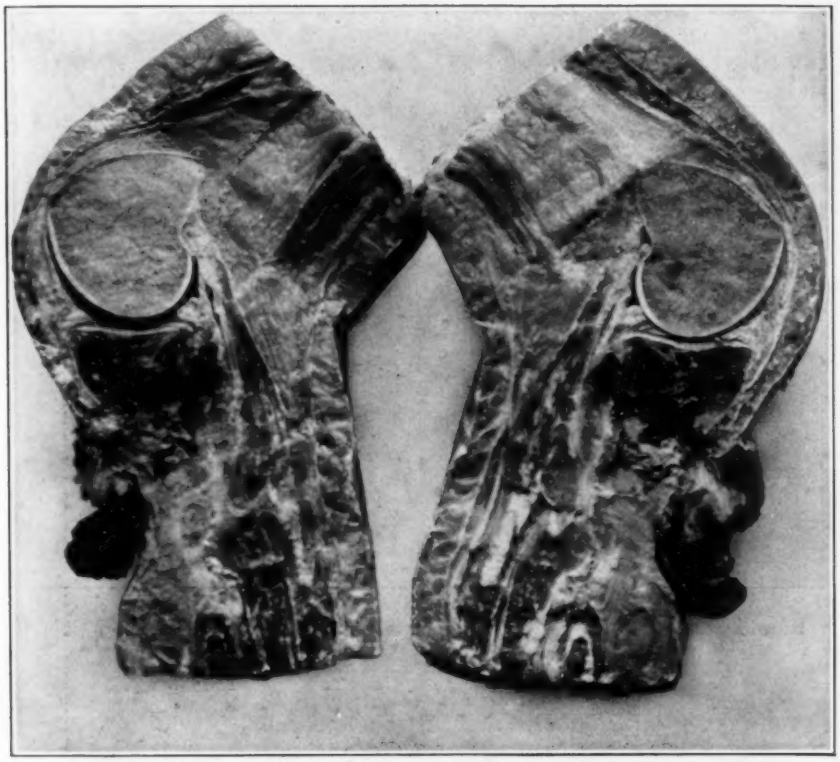

Fig. 24 (Case 34).—Section of limb shown in Figure 22. Note limitation of process by joint cartilage.

Fig. 25 (Case 34).—Recurrence in stump after amputation of limb shown in Figure 22.

had a pathologic fracture of the leg, at the site of which a tumor had developed and for which, one year later, the limb was amputated, with the diagnosis of giant cell tumor. Five years later, she died at the Presbyterian Hospital with a diagnosis of metastases in the chest. At

Fig. 26 (Case 34).—Section from primary tumor.

necropsy, the metastases were very difficult to interpret. The left lung showed a spindle cell type of growth, very cellular and free from giant cells; the right lung, growths of the border-line epulis type with many large giant cells (Fig. 21) containing from fifteen to twenty nuclei. It seems fair to infer from the history in this case that the original tumor, judging by its long course and its situation, was probably of the

epulis type. It is particularly unfortunate that the slides which would confirm the diagnosis of giant cell tumor in the original growth are lost, for if they were available, we would have complete for the first time the long sought sequence of primary giant cell bone tumor with death

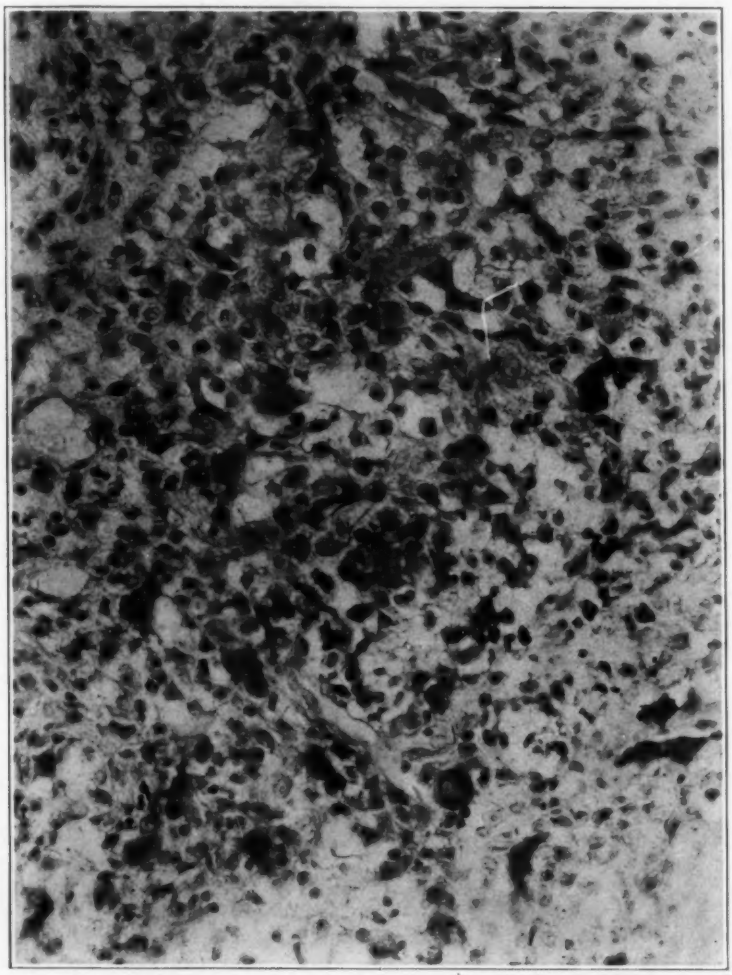

Fig. 27 (Case 34).—Section of recurrence from amputation stump.

by metastases, histologically similar to the primary tumor. We refuse, however, to be tempted into definite conclusoins in a case lacking sections of the primary tumor.

In the atypical type of giant cell tumors, Case 35 represents a borderline case, while Case 38 and Case 37 represent the other extreme, bordering very closely on the subperiosteal or medullary type of osteogenetic

sarcoma. Case 35 has certain features which we feel are of extreme importance. The patient was a woman of forty-five, who for eight years had been under observation with a diagnosis of giant cell sarcoma of the head of the tibia (Fig. 28) following an injury received four years previous to the time of examination. The increasing disability caused by the size of the tumor led her to consent to an operation. Her history, the roentgen-ray findings (Fig. 29) and physical signs were typical of giant cell tumor of the epulis type. A section removed for

Fig. 28 (Case 35).—Giant cell tumor of upper end of tibia of eight years duration. Curettage was followed by recurrence with malignant change.

diagnosis showed certain areas typical of giant cell tumor (Fig 31) and other areas almost entirely dominated by spindle cells. In spite of this, curettage was done, followed by cauterization. Three months later, there was a very rapidly growing local recurrence in the depths of the bone cavity, and besides this, out through the soft parts beyond the line of incision were several nodules (Fig. 30). The local recurrence and the nodules in the soft parts both showed a picture quite different from that of the primary tumor. The giant cell element had entirely disappeared, the mass being made up of very rapidly growing compactly arranged spindle cells (Fig. 32). The local recurrence had invaded the soft parts,

and this soft part extension, as well as a discrete nodule, showed, in addition to the foregoing picture, many small areas of newly formed bone. As a result of these findings, the limb was amputated at midthigh, and now, four years later, there is no evidence of recurrence or metasta-

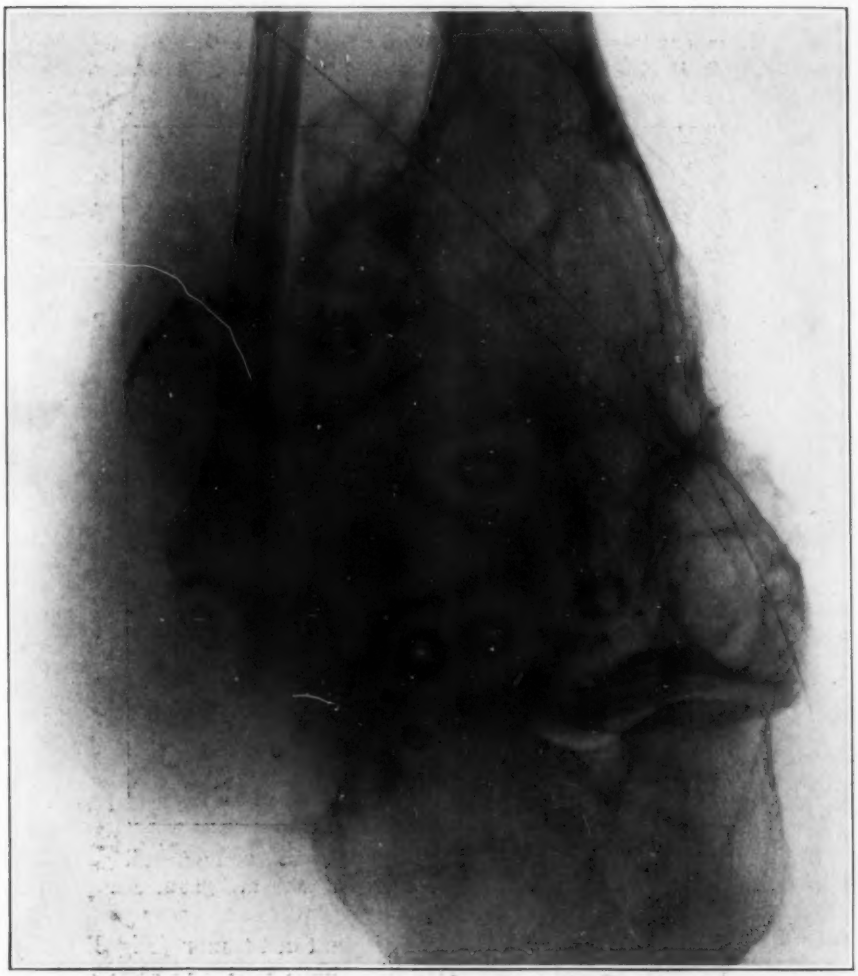

Fig. 29 (Case 35).—X-ray of giant cell tumor shown in Figure 28.

ses. We feel that this tumor was originally a straight-forward epulis type. Previous to admission, there had evidently been a rupture of the bony capsule. Whether or not this was connected with the delayed development of the malignant element, we cannot say. At the time of the first operation there already existed increased cellularity in the tumor, and it seems inconceivable that this could have been present for a

period of at least eight years. As a result of the curettage, this cellular element was apparently stimulated, accounting for the rapid recurrence. Amputation seems to have taken place in time to prevent metastases. Do all giant cell tumors have latent in them some malignant element, or is a giant cell tumor, as a whole, potentially malignant?

Fig. 30 (Case 35).—Section of limb after amputation. Note the cavity filled with a recurrence and the nodule beneath the patella.

We have grouped in this atypical epulis type Case 37 and Case 38 (Figs. 33, 34), although some might consider them types of medullary osteogenic sarcoma. Both these cases, however, were clinically diagnosed as giant cell tumor and, on section (Fig 35), showed many giant cells in a stroma of spindle and round cells. The malignant character of the

tumor was shown by a rapid recurrence after curettage, and amputation failed to prevent death by metastases.

In the present classification, there seems to be no heading other than giant cell tumor under which these two cases could be grouped, as they

Fig. 31 (Case 35).—Section from primary tumor, showing scattered giant cells.

clinically and histologically resemble the condition enough to be treated as such by curettage, and histologically, apparently because of the presence of a large number of giant cells, were diagnosed as cases of giant cell sarcoma. In addition, Case 38, a patient aged 10 years, would be

6. Bloodgood, J. C.: Ann. Surg. **69**:345-359 (April) 1919.

under 15 years of age, the low age limit for central osteogenic sarcoma set by Dr. Bloodgood.[6] These represent a class in regard to whose therapy surgeons have been led astray. Either they should be put under a separate classification, or the general rule made by Dr. Bloodgood that curettage as indicated in all these cases should be corrected. "If there

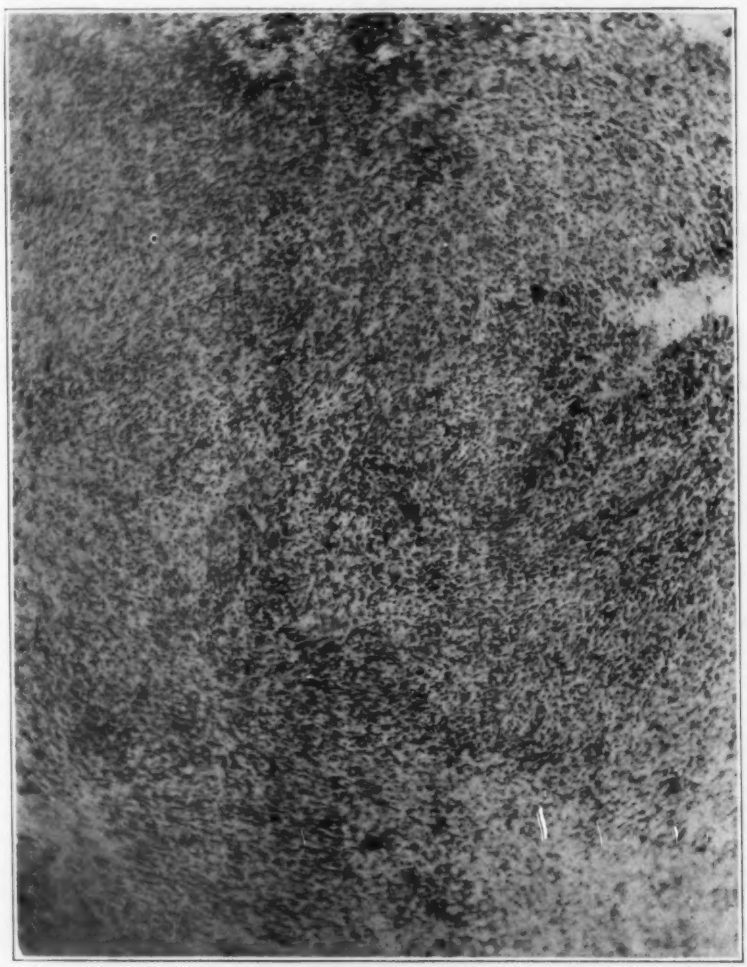

Fig. 32 (Case 35)—Section from recurrence of tumor shown in Figure 31. Note the disappearance of giant cells and the compact masses of spindle cells.

is any doubt as to diagnosis of malignancy, the lesion should be treated as if it were benign." We realize that Dr. Bloodgood made this remark to prevent unnecessary amputations, but, if any cases are amenable to cure by amputation, they are probably in this borderline group, and currettage seems doomed to failure.

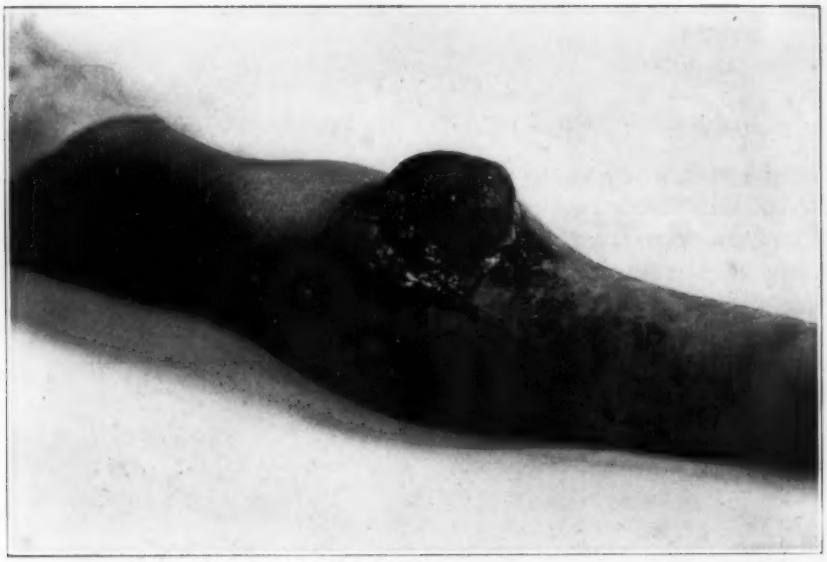

Fig. 33 (Case 38).—Recurrence of a giant cell tumor (?) after curettage.

Fig. 34 (Case 38).—Section through limb shown in Figure 33, showing origin of the growth in head of tibia. Death from metastases seven months after admission.

It has been of great interest to us in analyzing giant cell tumors in our series and in comparing the findings with those in the literature, to find our cases conforming to the description given by Nelaton [7] in 1863, and recently quoted by Ewing, rather than with the more didactic rules

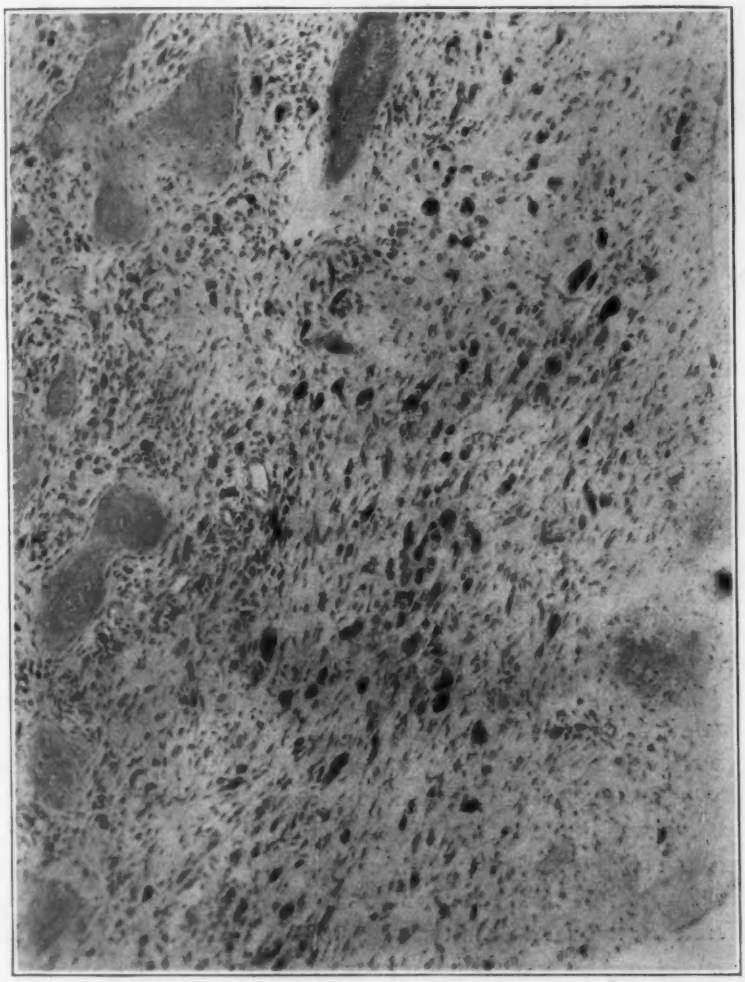

Fig. 35 (Case 38).—Section from the recurrent tumor shown in Figure 34. Note the new bone formation and the relatively small size of the giant cells.

laid down in the last fifteen years. Nelaton, sixty years ago, brought out the benign character of the typical giant cell tumor with the clinical and histologic description to which little has been added since. He also, however, noted the existence of a somewhat atypical giant cell tumor in

7. Nelaton: Tumeurs benignes des os, Paris, Gaz. d. Hop. 1863, p. 25.

which there was an increase in the proportion of spindle-shaped fibroblasts. He called attention to the possible malignancy of this class, and to the changed histologic picture with the increase in malignancy.

Of late years, there has been an attempt in this country to exclude giant cell tumors altogether from the class of sarcoma, and in this movement Bloodgood has been the leader. Our study in the present series has convinced us that this is unsound, and this impression has been emphasized after discussion with pathologists in other hospitals. A sufficiently large collection will probably show almost any degree of transition from the benign giant cell tumor to the very cellular spindle type of tumor in which the giant cells have disappeared. Is it fair to say that a giant cell tumor has metastasized as such, when metastasis shows only those latter spindle cell elements and not the primary type of tumor? Stone and Ewing [8] describe a giant cell tumor which showed increased malignancy in a recurrence after curettage, with disappearance of giant cells altogether from the last recurrence. Amputation failed to prevent death under a diagnosis of pulmonary metastases. This case had a very short history, there being only two years between the onset and the death of the patient. There was no necropsy which, of course, makes the diagnosis of pulmonary metastases speculative and leaves us in the dark as to the histology of the pulmonary lesion. This patient, at amputation, if one ignores the previous history, was suffering from a spindle cell sarcoma without the presence of giant cells. This case corresponds in the histologic changes to Case 35, in its early course; the latter however, had a much more typical eight-year course before operation. Both of these cases showed a change from comparatively benign to malignant tissue. Dr. Ewing thinks the "surgical insult" produces this change. Is it logical to blame any surgical procedure for the development of a malignant element in what is claimed to be absolutely benign tissue? If so, why is there not the same instance of this reaction taking place in some types of inflammatory tissue? Is it not fairer to consider, as apparently Nelaton did sixty years ago, that all giant cell tumors may contain elements of malignancy in varying degrees of latency? If this is understood, the use of the term sarcoma is of negligible importance.

### EWING'S TUMOR (PROBABLY ENDOTHELIOMA)

Under this heading we have included two cases (Table 7), in both of which the diagnosis on the slides had been confirmed by Dr. Ewing.

This is a very confusing group. Case 48, that of a tumor mass in the middle of the shaft of the femur, had a disarticulation at the hip

8. Stone, W. S., and Ewing, J.: An Unusual Alteration in the Natural History of a Giant Cell Tumor of Bone, Arch. Surg. **7**:280-296 (Sept.) 1923.

under the diagnosis of round cell sarcoma. This took place in 1909. In 1922, interest having been stimulated in the case, as the man was still free from recurrence or metastases, the slides (Figs. 36, 37) were reviewed and a diagnosis of endothelioma made by Dr. F. C. Wood

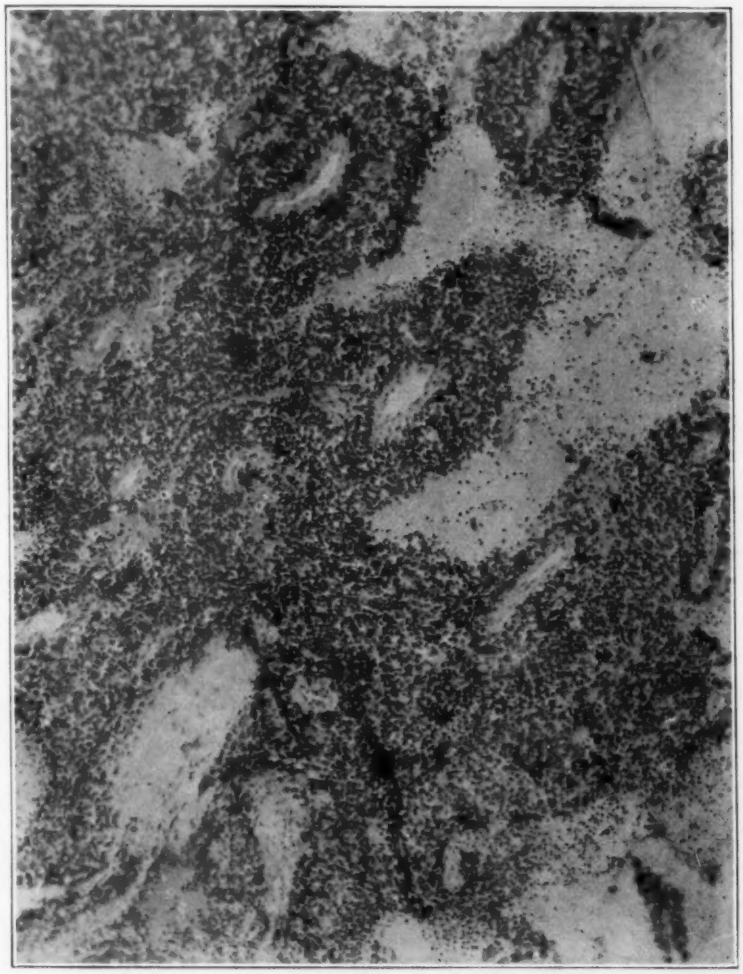

Fig. 36 (Case 48).—Ewing's tumor. Note dense masses of round cells containing many capillaries. This case is alive fourteen years after amputation.

and confirmed recently by Dr. Ewing. All the other members of the sarcoma committee being ignorant of the clinical course of this case, the slide was diagnosed as being from a highly malignant tumor. This patient was also treated with Coley's serum; a reference to this is made later under prognosis.

The second patient (Case 49) also had a mass in the left femur (Fig. 38). A resection was done under the diagnosis of endothelioma. One year later death occurred from general metastases in the skull and lung, in spite of treatment by both roentgen ray and radium. This

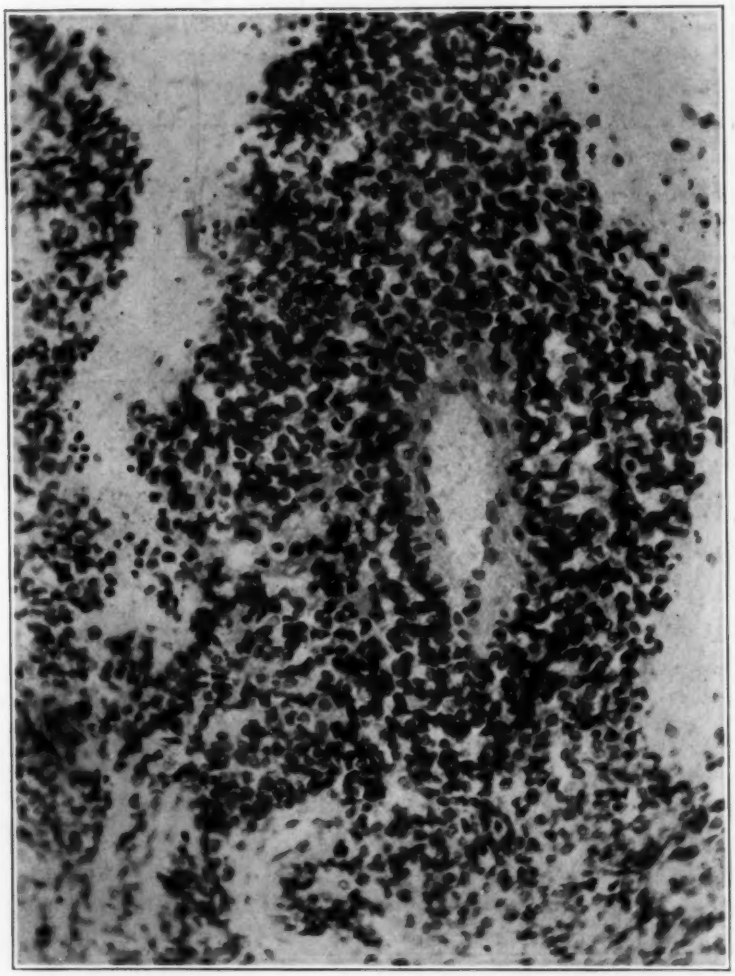

Fig. 37 (Case 48).—Higher power of section shown in Figure 36 showing the round cells grouped around a capillary. Note resemblance to Figure 11.

diagnosis was made at the time of the original operation by Dr. Ewing; theoretically, this case should have responded to roentgen-ray treatment.

In looking over our slides we found Case 24 (Fig. 11) a good example of what we used to call a small round cell sarcoma, the his-

tology of which is very similar to endothelioma. The tumor, however, was situated in the ileum and not in the shaft of a long bone.

We remain confused as to this group of cases. Is the diagnosis to be made on section alone, or on a combination of section, anatomic site

Fig. 38 (Case 49).—Ewing's tumor. Note the tendency of the round cells to form groups. This case died from metastases eleven months after resection

and clinical course? If on section alone, we have found that most pathologists would call this a round cell sarcoma. The anatomic site is not typical enough, and the clinical course requires hindsight rather than foresight. It may be that differentiation will ultimately be made by roentgen ray. It seems to be generally accepted that a definite pathologic

TABLE 7.—*Endothelioma*

| Case | Age | Sex | Admission Date | Site | Duration | Symptoms and Physical Signs | Clinical Diagnosis |
|---|---|---|---|---|---|---|---|
| 48 | 21 | M | 7/22/09 | Middle shaft of femur | 5 mo. | Pain in left thigh; loss of function; tumor mass, middle shaft of femur; bony hardness | Periostitis sarcoma |
| 49 | 30 | F | 11/ 9/21 | Upper end of left femur | 18 mo. | Pain; large, firm mass upper end of femur; tender | Sarcoma |

TABLE 8.—

| Case | Age | Sex | Admission Date | Site | Duration | Symptoms and Physical Signs | Clinical Diagnosis |
|---|---|---|---|---|---|---|---|
| 50 | 28 | M | 9/30/20 | Lower end of femur | 2 mo. | Small swelling, lower end of femur, gradually increasing in size; small tumor attached to lower end of femur, very hard | Osteomyelitis |

entity has been described by Dr. Ewing,[9] but we have found much difference of opinion as to the histogenesis of these tumors. Do endothelial cells represent a specialized group, and, if so, can any one say definitely that the round cells seen in these tumors are endothelial in origin?

Dr. William C. Clarke[10] of Columbia University, a few years ago, did considerable experimental work in an attempt to answer the first question. He found that ordinary connective tissue, in response to a non-irritating foreign body, would develop a layer of cells indistinguishable morphologically from endothelial cells. He came to the conclusion that endothelial cells were simply modified connective tissue cells.

The second question can only be answered by the possessor of that keen eye which will first perceive one of these cells leaving the lining of a blood vessel and starting its lawless career. The cell that makes up this tumor does not look like an endothelial cell and seems to have no specific staining properties. The sections of the tumor do not resemble in any characteristic way those tumors of the dura mater called endotheliomas by most pathologists. If they are not endothelial, what are they? We feel that it would be rather difficult to disprove that they are lymphatic in origin. They look more like lymphoid cells and they act more like lymphoid cells. We know that lymphosarcomas are exceed-

9. Ewing: Proceedings of N. Y. Path. Soc. **21**:17-25 (Jan.-May) 1921.

10. Clarke, W. C.: Experimental Mesothelium, Anatomical Record **10**:301, 1916.

*(Ewing's Tumor)*

| Roentgen-Ray Diagnosis | Pathology, Gross and Microscopic | Pathologic Diagnosis | Treatment | Recurrences and Follow-Up |
|---|---|---|---|---|
| Thickening of periosteum | Hard tumor mass surrounding bone (subperiosteal); large round cells; very vascular | 8/26/09, large cell sarcoma; 12/23/09, round cell sarcoma; 1922, endothelioma | First operation: tumor chiseled away from periosteum; 5 months later, disarticulation at hip joint for recurrence; Coley's serum | Recurred 4 months after first operation; following disarticulation, no recurrence after 13 years |
| None | Upper end of femur entirely destroyed; replaced by tumor; large round cells arranged in groups around a small channel | Endothelioma | Resection of femur; roentgen ray and radium | Died 11 months after admission; general metastasis in skull and lung |

*Parosteal Tumors*

| Roentgen-Ray Diagnosis | Pathology, Gross and Microscopic | Pathologic Diagnosis | Treatment | Recurrences and Follow-Up |
|---|---|---|---|---|
| Sarcoma | Amputation at hip; large tumor mass, involving muscle and attached to periosteum; dense connective tissue and areas of large interlacing spindle cells | Myosarcoma | Amputation at hip | No recurrence up to March, 1923 |

ingly variable in behavior, sometimes being highly malignant, and, in other cases, showing long periods of recession. Moreover, they seem, sometimes, to be peculiarly susceptible to roentgen-ray treatment. These physical and clinical similarities are offered simply as a demonstration of how difficult it is to disprove a preconceived theory in this field which has been so productive of didactic conclusions.

## PAROSTEAL TUMORS

Under parosteal we have placed one case (Table 8). The tumor was attached to the lower end of the femur, but its origin was probably in the soft parts. The pathologic diagnosis was myosarcoma. Amputation was done at the hip, and three years later there was no recurrence. Histologically, the tumor showed a malignant picture, but no other special features. In a tumor like this, it is almost impossible to differentiate between periosteal and parosteal.

## SUMMARY AND CONCLUSIONS

During the fourteen year period covered in this report, there were in the three hospitals represented many other cases of bone tumors not included in our series. It should be emphasized, however, that we included all cases having a pathologic specimen on file, that could be followed to date, and that could be classified logically under those headings which we have selected for study. This was the only basis of selection, and the findings are offered with no desire to support any preconceived theories.

The peculiar features of each group have already been discussed at some length, but for a consideration of the series as a whole, we feel justified in offering the following conclusions in regard to diagnosis, prognosis and therapy, in the rather puzzling clinical problems loosely called bone sarcoma.

Through history and physical examination it should be possible in most cases to exclude syphilis, tuberculosis, metastatic tumors, and osteomyelitis. Accurate differentiation depends upon roentgen-ray and pathologic section. It is essential, however, that the roentgenologist and the pathologist give an accurate description of the roentgenogram or the microscopic findings, and do not confine themselves to a diagnosis.

In 1918, Baetjer,[11] gave a splendid summary of the diagnostic possibilities and limitations of the roentgen ray in cases of bone tumors. From the roentgenogram the location of the bone tumor, whether it be central or periosteal, can be determined, and the presence or absence of a bony capsule, invasion of the soft parts, or of new bone formation can be detected. When the destructive process is slow enough to permit the formation of an expanding bony capsule, the prognosis is correspondingly improved. There is a rather characteristic picture in many giant cell tumors where the lesion shows an irregular, trabeculated formation of cortical bone somewhat similar to polycystic disease. These formations are almost always located in one of three places, at the head of the tibia, at the lower end of the femur, or at the lower end of the radius, extending to, but not through the cartilage. Ewing has found a characteristic roentgen-ray picture in diffuse, solitary endothelioma of bone.

Should a specimen of tissue section be removed in all cases of a suspected bone tumor? We feel very strongly that it should be a routine procedure. The only contraindication is the spreading of metastases, and our feeling is that only a hopelessly malignant type has this possibility. In placing a diagnosis on tissue section, the following points must always be borne in mind: (*a*) the tissue from one portion of the tumor may differ markedly from that of another; (*b*) the evidence of active mitoses varies with the time of fixation, and is found to some degree in all tumors; (*c*) microscopic reports should always indicate the histologic features of the tumor and not be confined to the individual diagnosis of the pathologist; (*d*) microscopic reports have great positive but little negative value; (*e*) prognosis based on a pathologic examination is, in many cases, speculative.

The findings in our series show great variation of interpretation of section, but the errors were usually due to an attempt to be too con-

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11. Baetjer, F. H.: Differential Diagnosis of Bone Tumors, Am. J. Roentgenol. **5**:260-264 (May) 1918.

clusive. The fundamental features of cellularity, morphology, mitoses, etc., are fixed and can be recorded accurately as such, thus avoiding sweeping conclusions.

After consideration of the clinical, roentgen-ray and pathologic findings, one should be able to arrive at a diagnosis of sarcoma in all cases, and in about 90 per cent. be able to distinguish between osteogenic, giant cell, and round cell or endothelial tumors. Further differentiation is bound to be arbitrary.

## TREATMENT

*Osteogenic Sarcoma.*—The very cellular type is uniformly fatal, and we found, in our series, no benefit obtained from the roentgen ray, radium, Coley's serum or amputation. Among patients with tumors of the sclerosing type four were alive two, four, six, and seven years after amputation or resection. It would seem that if metastases have already been dislodged, amputation is futile, and, if not, that resection suffices provided the soft parts have not been invaded.

*Giant Cell Sarcoma.*—If the histologic picture is typical, the treatment should be curettage and cauterization; if the case is at all doubtful, or if there is a recurrence, resection should occur; if the soft parts are involved, amputation should be performed. In the surgery of giant cell tumor, the marked susceptibility to infection after curettage should always be borne in mind and, when infection of the cavity has taken place, the strong probability of septic arthritis in the contiguous joint. We have no instance in our series of the disappearance of giant cell tumors after the use of the roentgen ray or radium. In tumors of the long bones, there are none of the restrictions on the free use of the roentgen ray or radium existing in the case of tumors of the trunk or head, and no contiguous viscera to be injured. In spite of this, our series of osteogenic and giant cell tumors show no evidence of benefit from the roentgen-ray or radium therapy, even where it has been applied in the depths of the bone cavity.

*Ewing's Tumor.*—One of our patients is alive fourteen years after disarticulation at the hip. This case was subjected to roentgen ray, Coley's serum and disarticulation. Which should be credited with the cure? Probably the answer lies in some peculiarity of the tumor itself.

## PROGNOSIS

*Osteogenic Sarcoma.*—This group as a whole showed 74 per cent. mortality. The mortality, as a rule, was in proportion to the malignant characteristics of the histologic picture, with the exception of the three cases classified as undifferentiated sarcoma. These possibly belong to the borderline giant cell group, as the patients are all alive, although histologically great cellularity was shown, and there was no attempt at

an adult form of tissue. The other cases of patients still living belong to the sclerosing group.

*Giant Cell Sarcoma.*—According to the cellularity of the stroma, a guess may be hazarded as to whether or not a giant cell tumor will recur. When recurrence had taken place, the histologic picture invariably showed a marked change, and we know of no guide as to the further course of the tumor. We know that in some cases invasion of the soft parts or of an amputation stump may take place, and in the borderline group we may get death from metastases. Where giant cell tumors recur, the recurrence is always of an altered character.

*Ewing's Tumor.*—This is a very variable tumor, with an uncertain prognosis leading to misinterpretation of the value of therapeutic agents. In all bone tumors, a definite prognosis, particularly as to time, should be avoided, as an accurate basis has not yet been established. Once metastases become manifest, however, a very rapid termination may be expected.

Throughout this review, both in the statistical analysis and the grouping of our conclusions, the classification of the committee on bone tumors has been faithfully adhered to, in order to preserve uniformity of discussion and to serve as a test of its practicability. A satisfactory classification must await that time when the etiology of neoplasms in general is less shrouded in mystery, and when the central registry shall have as a basis at least one thousand cases with complete clinical, pathologic and roentgen-ray data. Even without these as yet unavailable conditions, the classification submitted can obviously be much improved. In some divisions, there is an anatomic basis; in others, histologic, and in others, both. The result is that one case could logically be classified under more than one heading. For this reason, a word of warning must be given against statistics based on subdivisions.

Ewing's tumor is not yet established as a clinical entity. It would seem, therefore, wiser to give it a name, such as round cell sarcoma of the shaft, which would describe its outstanding features, rather than to give it the name of a controversial cell or of an individual.

# THE PATHOLOGY OF FAT EMBOLISM*

HARRY GAUSS, M.D.

DENVER

Fat embolism is an acute circulatory disturbance caused by trauma, manifested anatomically by the presence of fat globules within the vessels of the circulation and by certain secondary changes which these produce; clinically it is recognized by the presence of restlessness, dyspnea, delirium, coma and, frequently, death.

The cause of fat embolism is always trauma. It may have been such a trauma as to have caused a fracture of bones, a type which is most likely to produce fat embolism; or it may be the trauma which has caused a blunt injury to adipose tissue, or a rupture of certain viscera rich in fat, such as the liver, although this type of case is comparatively rare; it may occur in certain surgical procedures where considerable trauma is employed in the forcible correction of bones and joints such as sometimes happens in the operation of osteoclasis. Osteomyelitis has been mentioned as a cause of fat embolism, but the cases in the literature have not been authenticated.

The incidence of greatest frequency of fat embolism is in the third decade of life in men; it is then that men are exposed to the greatest occupational hazards, hence incurring the greatest amount of injury, with the possibility of fat embolism following injury.

The mechanism of fat embolism always results from mechanical factors, and the three distinct stages by which pathogenesis must proceed are: (1) the injury of adipose tissue, sufficient to produce a disorganization of the supporting fibrous tissue stroma to liberate free fat into an injured area; (2) the rupturing of a certain number of blood vessels, especially the veins within the abraided area; (3) the establishment of some mechanism which will cause the passage of the free fat into the open ends of the blood vessels.

In the vast majority of injuries to the body, in fact, in practically every case where there is hemorrhage into the tissues, the first two conditions are present, namely, the presence of free fat and the presence of the open ends of blood vessels; yet, fat embolism does not result because the third condition is absent. Those factors which favor the passage of fat into the open ends of blood vessels are not present. Normally, in an injury, where rupture of either large or small blood vessels takes place, these open ends do not remain open very long; for,

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* Read before the Medical Society of the City and County of Denver, March 1, 1924.

unless death follows, caused by hemorrhage, the blood vessels are closed by the normal process of repair. First, there is a collapse of the ends of the blood vessels; then, a clot forms at the torn end, effectively sealing it; later, organization and fibrosis takes place. A small amount of fat may have entered the blood vessel in the interval, but this is of no significance since the body is able to dispose of a small amount of fat. The four ways in which it may be disposed of are: (1) Passing into the general circulation, the fat may lodge within the glomeruli of the kidneys where,

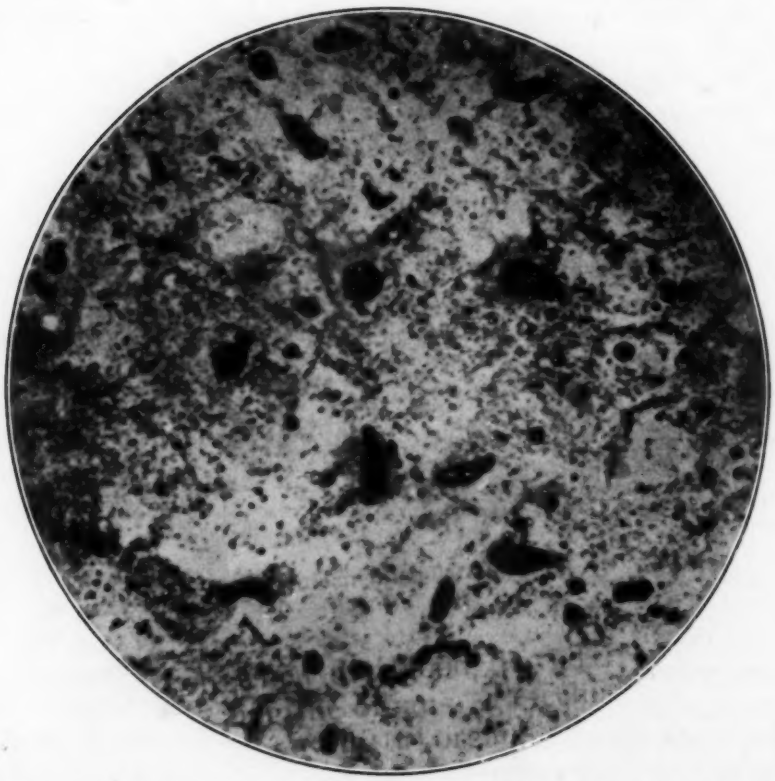

Fig. 1.—Photomicrograph (×60) of lung stained with sudan III and hematoxylin. Numerous fat emboli are seen within the blood vessels.

by capillary rupture or intercellular openings, it passes into the urine; (2) some may be excreted by the bile into the intestinal tract; (3) some may be taken up by the phagocytic endothelial cells and digested, and (4) the remainder may be emulsified by the mechanical action of the blood current, aided to some extent by the saponification action of the blood lipase and resorbed into the tissues.

The first two factors then, namely, the liberation of free fat and the opening of neighboring small blood vessels, are insufficient to cause fat

embolism; the third factor is also required. Some mechanism must be present for causing the passage of the free fat into the circulation. It is a matter of clinical experience that fat embolism commonly occurs in fractures and, most commonly, in fractures of the long bones; yet fractures of the long bones constitute only a very small per cent. of all injuries in which free fat is liberated in the neighborhood of broken blood vessels. There must be, then, some peculiar condition limited to injury by fractures which predisposes to fat embolism; this condition is the

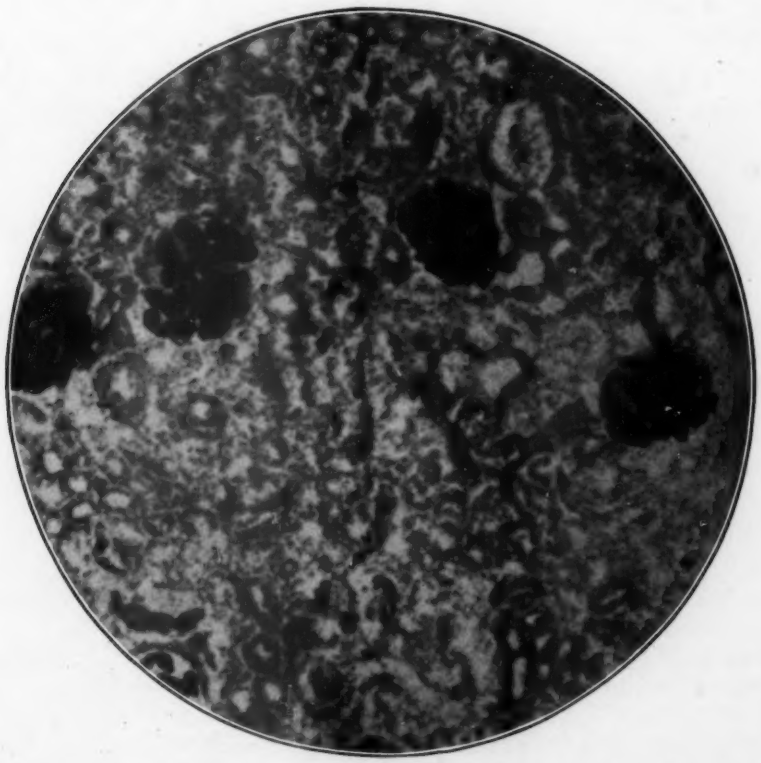

Fig. 2.—Photomicrograph (×60) of kidney stained with sudan III and hematoxylin. Fat emboli are present within the renal glomeruli.

presence of blood vessels within calcified tubules in the bones. These calcified tubules, osseous tubules, lying within the bones and holding the blood vessels fast to their walls, cannot collapse, and so these intra-osseous blood vessels are likewise prevented from collapsing. When a bone is broken sufficiently to open up some of these intra-osseous blood vessels, it is also broken sufficiently to cause a liberation of medullary fat, and, under these conditions, when the ends of the blood vessels are held open by their rigid perivascular sheaths, the third necessary condition is

provided for effecting the entry of fat globules into the circulation. Then the same physiologic forces that maintain the venous circulation operate to draw free fat into the wide open ends of the blood vessels, namely, the regional muscular movements, the negative venous pressure in the veins and other forces.

In this manner, considerable free fat enters the venous circulation; this is carried to the heart and thence to the lungs, where it lodges in the capillaries, forming effective emboli, completely blocking many of the smaller vessels, setting up a train of pathologic changes including hyperemia, edema, occlusion of the arterioles, petechial hemorrhages and, later, round cell infiltration. Clinically, these anatomic alterations become manifest as restlessness, rapid feeble pulse, pallor, cyanosis, dyspnea, increased respiratory rate, expectoration of bloody frothy

Fig. 3.—Photograph of transverse section of brain just anterior to the mammillary bodies. In the corpus callosum there are punctate hemorrhages.

sputum, together with physical signs of pulmonary edema. In certain cases the disease proceeds no farther than this stage. This is the respiratory type of fat embolism. The heart is unable to cope with the sudden pulmonary embarrassment, and the circulation begins to fall rapidly, death frequently following as a result of air hunger and asphyxia.

More often, however, the disease proceeds farther. The fat emboli pass through the capillaries of the lung, to return to the heart and enter the general circulation, which distributes them over the entire body. Entering the heart muscle through the coronary arteries, the emboli plug the small vessels, causing occlusion of the vessels with engorgement, hyperemia, hemorrhage, fragmentation, segmentation and fatty degeneration; this condition becomes manifest clinically by cardiac

Fig. 4.—Plot of brain showing its configuration and location of sections removed for study as described in a previous article by the writer. "A" represents the lateral view of the convex surface of the left cerebral hemisphere. "B" represents the convex surface from above. "C" represents the mesial surface of the left cerebral hemisphere. "D" represents the transverse section just anterior to the mammillary bodies. Seventeen blocks of tissue were removed for study from the following brain and cord areas: 1, spinal cord at the upper border of the decussation of the pyramidal tract; 2, vermis cerebelli; 3, right cerebellar cortex and dentate nucleus; 4, left cerebellar cortex and dentate nucleus; 5, left cerebral cortex, central sulcus, leg area, sensory and motor sides; 6, left cerebral cortex, triangular part of the inferior frontal gyrus, motor speech area; 7, left cerebral cortex, superior and middle temporal gyri, auditory area; 8, left cerebral cortex, calcarine fissure, including portions of the cuneus and lingual gyri, vision area; 9, right cerebral cortex, leg area, central sulcus, including sensory and motor sides; 10, left cerebral cortex, superior and middle frontal gyri, association area; 11, left cerebral cortex, gyrus angularis, inferior parietal lobule, association area; 12, left cerebral cortex, central sulcus, arm area including sensory and motor sides; 13, body of corpus callosum near the middle of its anteroposterior extent, right side; 14, right ventral limb of internal capsule including portions of the globus pallidus and thalamus. This and the following three sections were taken from the transverse section of the brain just anterior to the mammillary bodies; 15, right dorsal limb of internal capsule including portions of the putamen and caudate nucleus; 16, left ventral limb of the internal capsule, including portions of the globus pallidus and thalamus; 17, left dorsal limb of internal capsule, including portions of the putamen and caudate nucleus.

embarrassment such as a rapid feeble pulse, falling blood pressure and dyspnea; obtaining entrance into the kidneys, the emboli fill the glomeruli, inciting a beginning glomerulonephritis resulting in hyperemia, hemorrhage, fatty degeneration and some round cell infiltration. Clinically, this becomes manifest by lipuria and sometimes by the presence of albumin and casts in the urine. In a similar manner, the fat emboli enter every other organ in the body, producing more or less parenchymatous changes and some functional disturbance. But the organ or rather the

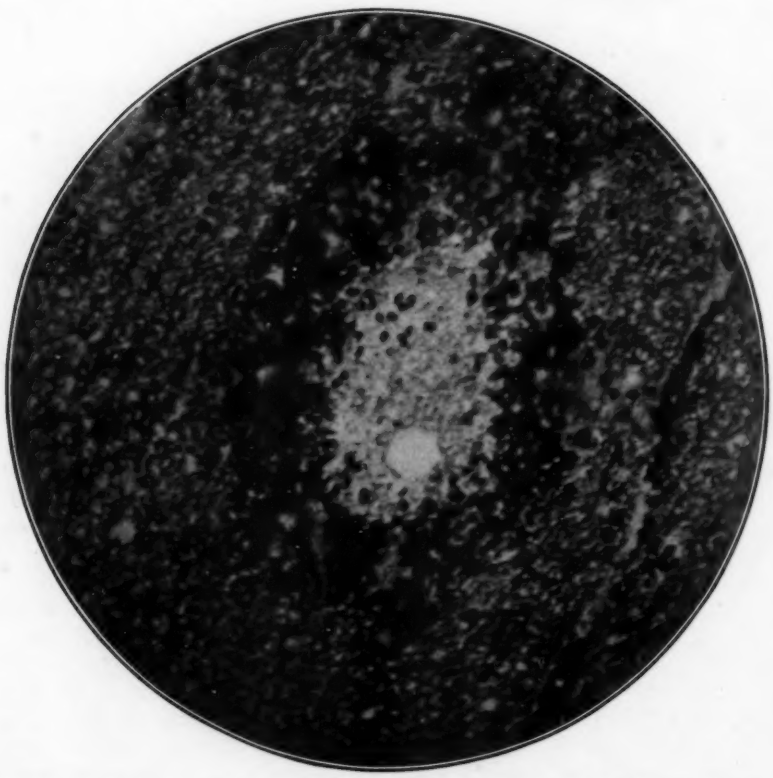

Fig. 5.—Photomicrograph (×230), showing circular hemorrhage in the leg area of the precentral gyrus, right hemisphere; Weigert myelin sheath stain. Within the hemorrhage, the myelin sheaths are entirely destroyed.

system showing the greatest anatomic disturbance and corresponding functional manifestations is the central nervous system. Every part of the central nervous system appears to be involved in this process. Sections taken through all levels of the spinal cord, through the spinal root nerves, through the cerebellum, vermis, dentate nucleus, from all portions of the cerebral cortex, the sensory and motor areas, motor speech area, auditory area, association area, from all parts of the internal capsule and

the adjacent nuclei, corpus callosum, and corona radiata, all manifest fat emboli, occluding the capillaries, edema, hemorrhage, and round cell infiltration; now, in addition, there are seen multiple small areas of focal necrosis in which there is a total destruction of the myelin sheaths and a loss of the axones and neurofibrils. The ganglionic cells show degenerative changes including shifting of the ganglionic nucleus to an eccentric position, swelling of the cell with loss of its characteristic polygonal shape, swelling and distortion of the nuclei, then karyolysis

Fig. 6.—Photomicrograph (×110), showing fat emboli in the cerebellar cortex; osmic acid stain.

and cytolysis. Finally, after disintegration of the cell nucleus, there are seen degenerative changes in the axones and neurofibrils. In every ascending and descending tract of the spinal cord and white substance of the brain, brain-stem and cerebellum, these degenerative changes have been observed.

Clinically, these changes are manifested, first, by restlessness, which becomes progressive in its intensity, does not respond to any form of medication, but passes into a delirium which is later so violent that

mechanical restraints are usually necessary as the only possible means of controlling the patient. This delirium is sometimes accompanied by vomiting, trismus, convulsions, high fever; then the delirium begins to subside, and the patient passes into a somnolent state followed by stupor; sometimes, all the pulmonary and cardiac symptoms are present. Finally coma and Cheynne-Stokes respiration set in, conditions which are usually the forerunners of the fatal end. This is the cerebral type of fat embolism.

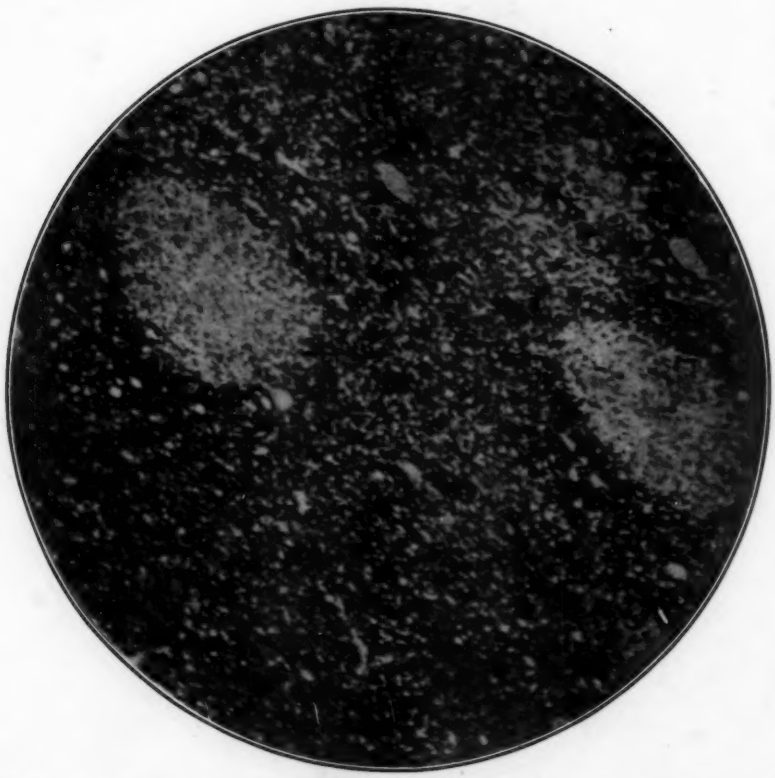

Fig. 7.—Photomicrograph (×60), showing multiple foci of necrosis in the arm area of the precentral gyrus, left hemisphere; Weigert myelin sheath stain. In the light staining areas there is a loss of most of the myelin sheaths.

About ten years ago [1] we became interested in the pathology of fat embolism at the suggestion of the coroner physician of Chicago, because of certain medicolegal aspects growing out of fracture cases. At that

1. LeCount, E. R., and Gauss, Harry: A Study of Fat Embolism Associated with Fractures, Trans. Chicago Path. Soc. **9**:251, 1915. Gauss, Harry: Studies in Cerebral Fat Embolism with Reference to the Pathology of Delirium and Coma, Arch. Int. Med. **18**:76 (July) 1916.

time, ten years ago, this country was in its preprohibition era, and there existed the custom among a large number of people in Chicago of including the whisky bottle in all first aid outfits, so that many patients were brought into hospitals following accidents with the odor of alcohol on their breath. Later, when these victims of accidents developed delirium the diagnosis was frequently delirium tremens and, when they died, the death certificates were signed accordingly. People who die of delirium tremens are generally regarded as dying as a result of their

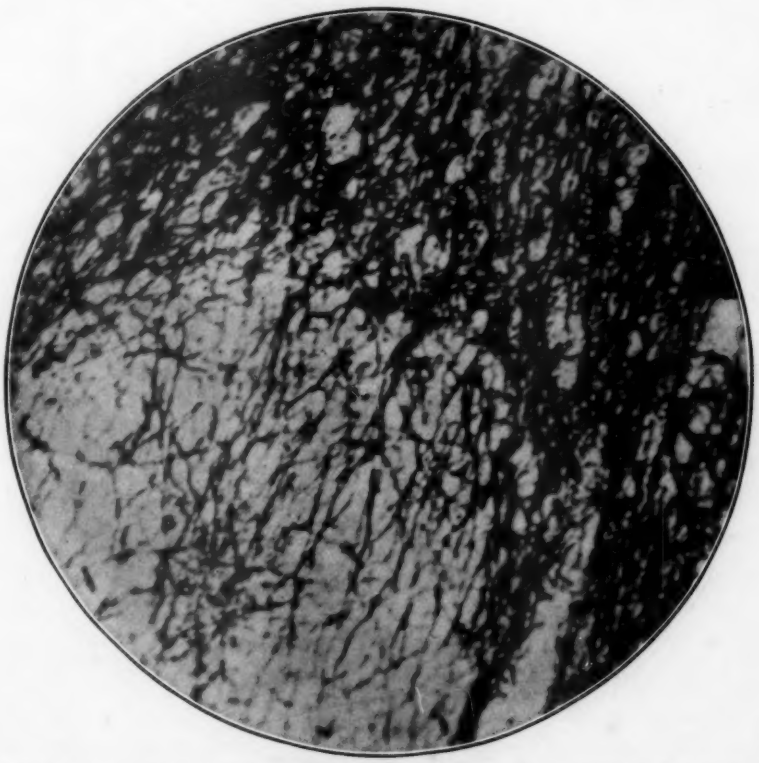

Fig. 8.—Photomicrograph (×325), showing focal necrosis in the motor speech area from the triangular part of the inferior frontal gyrus of the left hemisphere. Silver pyridin preparation for neurofibrils. Within the focus four-fifths of the axones have been destroyed.

own misconduct, and thus claims arising from a death attributed to delirium tremens get scant sympathy either in court or out of court.

It was largely because of these medicolegal complications that we undertook the study of the pathology of fat embolism to see if we could determine the pathologic basis for the delirium and subsequent coma that sometimes follow fractures, particularly the fractures of long bones. In our studies, it will be recalled that we found every part of the central

nervous system from the cerebral cortex down to the peripheral nerves containing capillaries plugged with emboli; edema, congestion, petechial hemorrhages, small areas of round cell infiltration, degeneration of ganglionic cells where such cells were present and multiple small areas of focal necrosis, within which there was a destruction of the myelin sheaths and neurofibrils.

If we now examine the conduction pathways of the central nervous system, selecting representative ascending and descending tracts, and

Fig. 9.—Photomicrograph (×275), showing changes in the denate nucleus of the cerebellum; Weigert myelin sheath stain. In the right lower quadrant, there is an area of focal necrosis in which most of the myelin sheaths are lost. Two ganglionic cells immediately adjacent to it have undergone karyolysis and partial cytolysis. A cell in the left lower quadrant has an eccentric nucleus; the dark staining cell in the center of the field is normal and the cell above it has lost its characteristic morphology and stains lightly.

inquiring into the influence exerted by these anatomic alterations on the functional activity of both these systems, it becomes evident that there is a marked interference with the passage of both afferent and efferent normal impulse; there is produced by the pathologic state an initiation of

an avalanche of psychomotor and psychosensory impulses causing marked clinical manifestations and ultimately leading to a complete disorganization of the central nervous system.

When the ascending exteroceptive pathway conveying impulses of pain, touch, temperature and pressure is examined, this tract is seen to consist of three neurons. Neuron 1 enters the dorsal spinal root and terminates in the grey matter of the dorsal column of the same

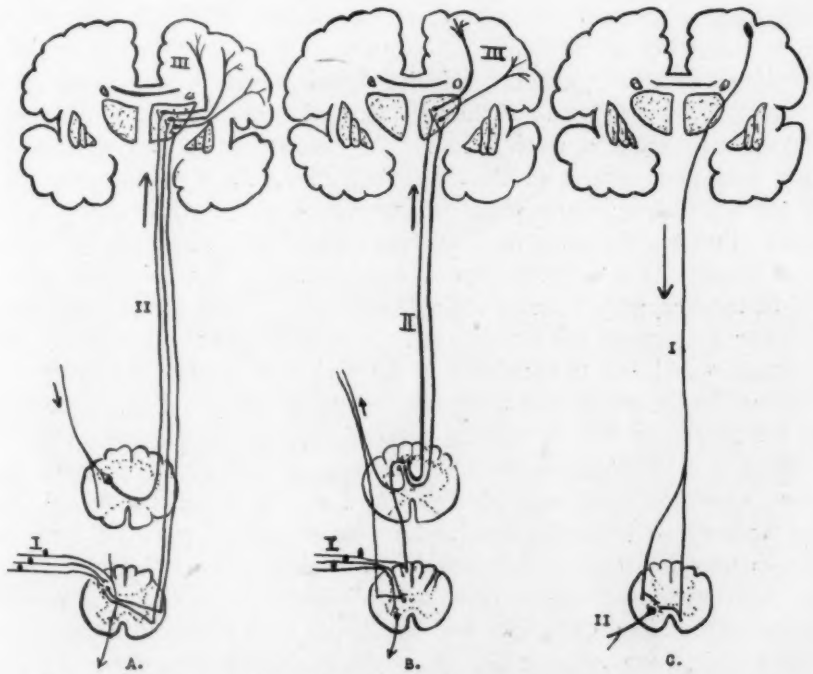


Fig. 10.—Representation of the ascending and descending pathways of the central nervous system by their constituent neurons. *A*, ascending exteroceptive tracts for touch pain, temperature and pressure: Neuron I, dorsal root to end in gray matter dorsal column, same side; Neuron II, crosses at spinal level and ascends in spinal lemniscus to thalamus; Neuron III, from thalamus through internal capsule and corona radiata to cortex. *B*, ascending tracts, proprioceptive muscle and joint sense: Neuron I, dorsal root to end in nuclei of fasciculi gracilis et cuneati; Neuron II, medial lemniscus from lower nuclei to thalamus opposite side; Neuron III, from the thalamus through internal capsule and corona radiata to cortex. *C*, descending tracts, motor control: Neuron I, cortex via corona radiata internal capsule, thalamus direct pyramidal and crossed pyramidal tracts to spinal opposite side of cortex; Neuron II, anterior gray horn to periphery via anterior spinal root. At the level of each of these constituent neurons, as well as in the cortical gray matter of the cerebrum, there were found fat emboli occluding the capillaries, and also the secondary changes including the multiple areas of necrosis. It is these pathologic conditions that cause the delirium and coma, and sometimes the death of patients who develop fat embolism following fractures.

side. Neuron 2 begins here, crosses over and ascends in the spinal lemniscus to end in the thalamus. Neuron 3 begins here, then passes from the thalamus through the internal capsules, and through the corona radiata to the sensory part of the cortex in the postcentral gyrus. At every one of these levels, emboli and degenerative changes were found. The ascending proprioceptive pathways of muscle and joint sense likewise consist of three neurons. Neuron 1 enters the dorsal root and ascends in fasciculus gracilis or cumeatus to end in the nuclei of these columns. Neuron 2 starts here, crosses in the medial lemniscus and ascends to end in the thalamus. Neuron 3 starts here, passes through the internal capsule, and through the corona radiata to end in the postcentral sensory gyrus. At every one of these levels, emboli and degenerative changes were found. The descending pathways of voluntary muscular control in their simplest case consist of two neurons. Neuron 1 starts in the precentral gyrus of the cortical motor area, passes through the corona radiata, internal capsule, and thalamus; then it descends in the direct pyramidal and crossed pyramidal tracts to end in the anterior grey horn on the side opposite to its origin in the cortex. Neuron 2 starts in the anterior grey horn and passes out through the anterior spinal root to terminate in the peripheral tissue. At every one of these levels, emboli and degenerative changes were found. Likewise, in the ascending and descending cerebellar pathways, as well as in the cerebellar nuclei, the degenerative changes were found. Not only is every conduction pathway of the central nervous system involved, but the sensory postcentral gyrus and motor precentral gyrus, the motor speech area in Broca's convolution, the auditory temporal area as well as the several association areas are all found to contain fat emboli plugging the blood capillaries and impinging against the adjacent nerve tissue, and all are accompanied by secondary degenerative changes.

It is a fact of physiology that stimulation of either the sensory cortical area of the brain or any of its conduction paths initiates a psychosensory impulse, and that stimulation of the motor cortical area of the brain or any of its conduction paths initiates a psychomotor impulse. In fat embolism, myriads of small emboli enter the central nervous system and impinge against the adjacent nerve tissue from which they are separated only by a thin endothelial wall and so mechanically initiate stimuli giving rise to psychomotor and psychosensory impulses. Myriads of these impulses are initiated, for no apparent reason and without coordination of any kind; the clinical result is delirium.

At first, these emboli mechanically irritate the adjacent nerve tissue, but soon they cut off the blood supply and interfere with the nutrition of the cell. Later, as the secondary changes of edema, hemorrhage and focal necrosis appear, these nerve cells and their axons undergo necrosis and finally death; thus is instituted a disorganization of the central

nervous system leading to its complete functional collapse. This law is a reversal of the law of evolution. According to the law of dissolution, the highest nervous processes, since they are the latest to form and consequently the least organized, are the first to disappear in a manner similar to the operation of anesthesia; the lowest nervous processes, since they are the longest and most completely organized, are the last to disappear. Coma is a late manifestation of the law of dissolution, which is set in motion in this condition by the complete disorganization of the central nervous system as a result of the multiple areas of focal necrosis.

This, I believe, to be the basis of the pathology of fat embolism, especially of the delirium and coma that sometimes follow fractures.

# UTERUS DIDELPHYS WITH TORSION OF THE PREGNANT RIGHT UTERUS

HARRY W. HORN

WICHITA, KAN.

The occurence of uterus didelphys in itself is far from rare. This is also true of the frequency of pregnancy in one or both halves of a double uterus, as the condition is most frequently observed when associated with pregnancy and not often recognized unless accidentally discovered when the abdomen is invaded surgically or when this condition is discovered during pregnancy. However, torsion of the pregnant half of the uterus bringing about such severe circulatory changes that gangrene of the affected part ensues, such an effect as the changes occurring in any pedunculated tumor with twisted pedicle may produce, is extremely rare. A careful investigation of the literature thus far has not revealed a single recorded instance of this complication of pregnancy in the case of a uterus didelphys, torsion of the pregnant half.

A brief resumé of the embryology will be given before recording the history and operative findings of this patient. Malformations of the uterus and its appendages are not uncommon. These organs embryologically are developed from the ducts of Müller. Between the fifth and eighth weeks of embryonal life, the anterior ends of Müller's ducts begin to rotate about the Wolffian ducts, finally coming to lie in front and to the median side of the latter. At about the eighth week of embryonal life, the ducts, originally two separate tubes, begin to fuse. The upper or proximal ends remain separate and ununited and become the fallopian tubes or oviducts. The middle thirds undergo fusion and form the uterus. The distal ends coalesce to form the vagina. This process of fusion transforming the double duct into a single uterus and vagina goes on gradually. These segments coalesce externally, their united median walls still forming a septum. This septum gradually disappears and about the twelfth week of fetal life, the uterus and vagina become single organs.

The arrest of or interference with the continuation of this process leads to some form of malformation or duplexity of the uterus or vagina or both. The character or type of malformation or duplexity depends upon the time in the embryological development when the arrest in the fusion of the fully developed ducts takes place. Malformations, as a result of imperfect development of one or both Müllerian ducts or their incomplete fusion, may be grouped into two divisions. In the first group may be placed those which present external evidences that complete

fusion of the fully developed ducts did not take place, and in the second may be placed those in which there is no external evidence that complete fusion of the fully developed ducts did not take place.

### TYPES OF MALFORMATION

GROUP 1.—*Uterus bicornis.*—In this type of malformation, the upper parts (variable in length) of the middle third of the Müllerian ducts do not fuse, each upper part thus forming a distinct horn. The remaining lower portion of the middle third always fuses externally, while the septum internally may or may not become entirely obliterated, i. e., that portion of the uterus below the diverging horns may or may not be divided into two separate cavities by a septum. If it is divided by a septum and the septum extends to the external os, it is termed uterus bicornis bicollis. If the septum becomes obliterated or fails to extend to the external os, we term it uterus bicornis unicollis.

*Uterus didelphys.*—In this type, the middle third of each duct, which, by the process of fusion, normally develops into a single uterus, remains separate. Each half develops into a distinct organ with a single tube and ovary. There is total absence of fusion, both uteri including their cervices are entirely separate from each other. They are usually uniform in size.

GROUP 2.—*Uterus bilocularis or uterus septus.*—Those cases which present no external evidence that complete fusion of the Müllerian ducts has not taken place belong to the second group. Externally, there has been complete fusion of the ducts. The external contour of the fundus is normal. Internally, the obliteration of the septum has not taken place, and there is an internal division. This division may be partial or complete. It is complete when it extends to the external os, and it is then termed uterus septus bicollis; when it does not extend to the external os it is termed uterus septus unicollis.

In all three of the types so briefly described the distal third of Müller's ducts may or may not have undergone fusion so that in any of them may be found a single or a double vagina.

### REPORT OF CASE

*History.*—Mrs. S. J., aged 20, primipara, was married Feb. 10, 1923. Her last menstrual period occurred July 29, 1923. Motion was first observed December 10, and continued without interruption up to the day of the termination of pregnancy. Except for slight nausea during the earlier days and for occasional vague pains referred to the right half of the abdomen during the fifth month, the patient did not experience any unusual symptoms.

On September 22, she consulted her family physician, Dr. Chas. Caswell, for the first time. His examination at that time brought out the following: except

for the diseases of childhood, the patient has enjoyed good health. She first menstruated at the age of 12, and the periods were of the twenty-eight day type; lasting from three to four days; very little pain accompanied menstruation, and there was no intermenstrual discharge. The patient knew very little concerning her family history except that she was one of seven children and that two sets of twins preceded her in order of birth.

*Examination.*—The pelvic examination revealed no deviation from the normal. The abdomen and chest were normal; the urine was normal; hemoglobin, 95; red blood cells, 4,780,000; no white count was made, and the blood pressure was

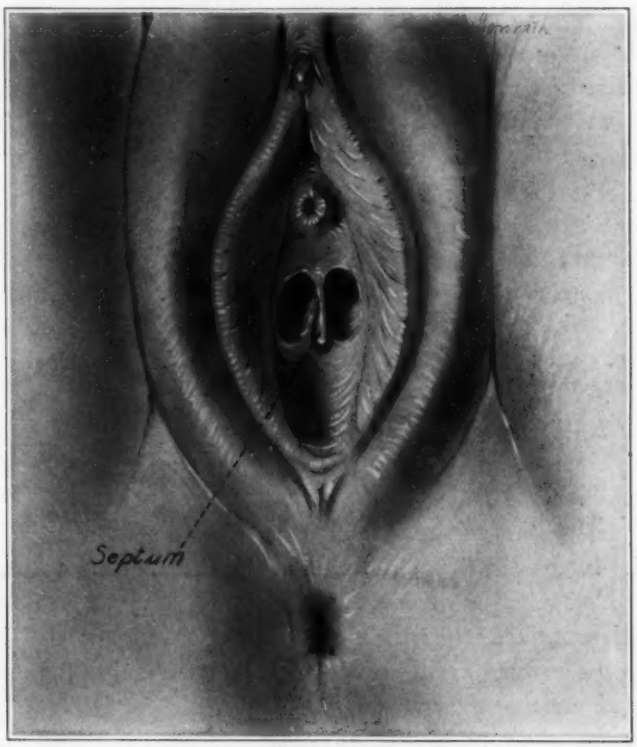


Fig. 1.—External appearance of double vagina, showing divided septum.

120. The pelvic measurements were: intercristal, 24 cm.; interspinal, 26 cm.; external conjugate, 19 cm.; diagonal conjugate, 12.5 cm.

Monthly examinations were made from this time on with no unusual developments. Jan. 7, 1924, was the time of her final visit to her physician's office, when she informed him of a mild, indefinite pain in the right half of the abdomen, felt during the preceding month. The fundus uteri at this time reached the level of the umbilicus. The general condition of the patient was excellent.

*Further Developments.*—January 8, at 6:30 a. m. the patient was aroused from a sound sleep by excruciating and continuous pain in the lower half of the abdomen. At no time was this pain intermittent in type. There was no flow of

blood from the vagina. The pain persisted without relief and at 8:00 a. m., Dr. Caswell visited her. Her temperature was 97, pulse 84, and she presented the facies of one in shock. There was no vomiting. The fundus of the uterus could be made out at the level of the umbilicus, and there were no uterine contractions. The uterine wall was firm, somewhat tender and irregular in outline, corresponding in size to a uterus between the fifth and sixth month of pregnancy. Morphin, ¼ grain (0.016 gm.) with atropin, grain 1/150, was given hypodermically for pain.

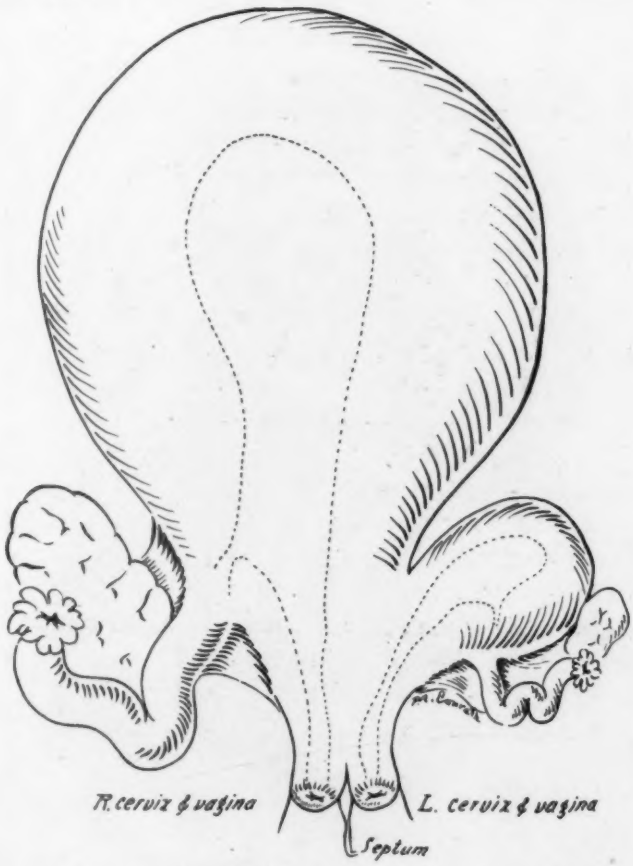


Fig. 2.—Uterine bodies, cervices, and double vagina.

Two hours later, her physician observed her again. Her temperature was 97; pulse 95. There was no change in the level of the uterus. The skin was cool, and her facies remained as before. The blood examination made at this time revealed a hemoglobin of 80; red blood cells, 3,230,000; white cells, 17,600.

At 1:30 p. m., the writer saw her in consultation with her physician. The pulse and temperature remained as last observed. The blood pressure was: systolic, 120; diastolic, 46; the pulse was soft in quality. The facies was suggestive of shock though the pulse was not rapid. The pain was less severe as a result of morphin.

The fundus of the uterus was now found in the left hypochondriac region. This change in the height of the uterus had taken place in two and one-half hours. The abdomen was tender, and the uterine wall was soft and flabby in consistency. Rectal examination was very unsatisfactory because of the abdominal condition. The cervix could be felt through the rectum and gave one the impression of a dilating cervix. No vaginal examination was made.

Immediate operation was advised, after a probable diagnosis of premature detachment of the placenta was made. The patient was immediately removed to the Wesley Hospital, where the operation was performed.

Fig. 3.—Illustration showing clockwise torsion of the right uterus (gangrenous) on its lower segment. The tube and ovary shown as being on the left side are the right tube and ovary, transposed through the torsion of the uterus. Each uterus possessed but one tube and one ovary.

*Operation.*—The operation took place, Jan. 8, 1924, at 4:00 p. m. Gas-oxygen anesthesia was administered by Dr. F. H. Slayton. The assistants were Dr. A. L. Crittenden and Dr. Chas. Rombold. A median incision, the center of which was opposite the umbilicus, was made. Upon incising the peritoneum, some clear fluid escaped. The presenting surface of the uterus was black and the uterine wall had lost its tonicity. A longitudinal incision was made into

the presenting surface of the uterus. The cavity of the uterus contained about 500 c.c. of fluid blood. The placenta was found completely detached from the wall of the uterus. The placenta and unruptured sac were delivered with ease, and the uterus was sutured. Hot saline packs were used to stimulate the uterine circulation, but to no avail. Because of the gangrenous condition of the uterine wall, hysterectomy seemed advisable. The highly placed incision was extended to the symphysis, and it was then discovered that the pregnant uterus was twisted clockwise to the left 180 degrees on its lower cervical segment. On the left side of the uterus, was located the right tube and ovary, which had

Fig. 4.—Pregnant right uterus as they appeared after relieving the torsion of the right uterus. The black line divides the level through which supravaginal hysterectomy was performed.

undergone changes similar to the circulatory changes in the uterine wall. On relieving the torsion of the uterus, it was found that the incision had been made in the posterior uterine wall. There was no left tube and ovary, but low down in the left pelvis was found the left half of a double uterus, of which the pregnant uterus was the right half (uterus didelphys).

A supravaginal hysterectomy was performed on the right side at the site noted in the accompanying drawing. The left half was the size of a pregnant uterus between the first and second months. It was firm in consistency and symmetrical

in contour. There was the usual tube and ovary on the left side, but none on the right side. The incision in the abdominal wall was closed after soft rubber drainage was provided.

There was no evidence of pregnancy in the left half. There were two separate cervices with separate canals. The vagina was double, with the usual longitudinal septum. Ten days later, the vaginal septum was removed under local anesthesia. There was no evidence of a decidual membrane in the non-gravid side. The patient made an uneventful recovery.

The fetus corresponded in size and appearance to that of one between five and six months in age. It was a male and presented no anatomical defects.

February 19, the patient resumed her menstrual periods, flowing profusely for five days and experiencing severe pain in the left lower abdominal quadrant.

## SUMMARY

Malformations of the uterus associated with pregnancy are not rare. Many cases have found their way into the literature. Torsion of the gravid side of a uterus didelphys is extremely unusual and in the case recorded here, there was no history of any exciting or predisposing cause.

# MUSCLE TEARS*

A. GOTTLIEB, M.D.

LOS ANGELES

Rupture of muscles and avulsion of their attachments have been liberally dealt with in medical literature and have received consideration in regard to diagnosis, prognosis and treatment. Muscle tears, i. e., injuries of the muscle substance with little disturbance of the continuity of the muscle, have been treated very sparingly; they have either remained unrecognized or have been taken lightly and disregarded. They are more common than reports indicate and deserve attention.

## ETIOLOGY

Muscle tears occur more frequently in man than in woman, apparently because the occupations of men cause them to be more exposed to injuries. They are more prone to happen in persons above 40 years of age, when the body elasticity and resiliency have lessened. Certain muscles tear in the course of definite occupations, viz.: the adductors of the thigh in horseriders, the neck muscles in load carriers, the calf group of muscles in mountain climbers, runners and tennis players. Lawn tennis leg was the name given by Küttner [1] to calf muscle tears.

The mechanism responsible for the production of a tear cannot always be established with ease. A tear occurs in a contracted muscle when a sudden, poorly coordinated movement is executed in a moment of unawareness or surprise; it results from an unconscious act of protection against a threatening danger. The rider's thigh may best serve as an example. In horseback riding the adductors of the thigh are firmly contracted to hold the knees against the saddle; an unexpected movement of the horse may partially unseat the rider and throw his legs forcibly against the side of the saddle. Then the contracted adductors snap like a taut bowstring. Since muscles will tear more readily if tired from overuse the state of fatigue of the muscle at the time of the accident, as well as the general health of the individual, must be considered. Sufferers from syphilis, arteriosclerosis, arthritis deformans, acute infectious diseases, alcoholism and other constitutional defects are more apt to sustain this as well as any other injury.

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* Read before the Industrial Accident Section of the Los Angeles County Medical Society, March 13, 1924.

1. Küttner, H.: Lawn Tennis Leg, Deutsch. med. Wchnschr. **48**:412 (March 31) 1922.

SYMPTOMATOLOGY AND TREATMENT

The onset is sudden, accompanied by a severe sharp pain, as if a knife had pierced the region. As a rule, the individual instantly stops all activity in the injured part. An audible and perceptible crack may accompany the tear. The objective signs vary with the location and the degree of the tear: there will always be tenderness on pressure over the site of the tear and increase of pain if the motions executed separate the torn fibers. A groove or a depression in the muscle substance may not always be palpable, and ecchymosis from capillary hemorrhage may be present if the tear is superficial.

In the order of frequency, as Grassheim [2] found in more than 500 cases, as confirmed by Leusden [3] and evidenced by my own twenty-eight cases of muscle tears, I will briefly describe injuries located in various regions of the body.

1. Tears of the spinal muscles or fasciae, known by the misnomer lumbago, are unquestionably of frequent occurrence. Characteristic objective findings are absent, as a rule, and we are guided in the diagnosis by the history of the accident and the exclusion of other pathologic lesions, such as fractures, ostearthritis, myositis from exposure to cold, postural backaches and congenital anomalies of the pelvic girdle and lower spine. Previous history may exclude some of these, while a complete examination, including a roentgenogram, will eliminate the others. The rapidity of recovery from the alleged injury may lead to the diagnosis of muscle tear because minor tears will heal in from ten to fourteen days; if recovery is delayed longer, and other possibilities are eliminated, there will be found an area of swelling due to diffuse bleeding and a cleft in the muscle substance which will be proof of more extensive muscle and fascial tears.

The treatment in recent cases consists in exposure to radiant light and heat from a high power incandescent lamp or from a hood lined with numerous light bulbs. This is followed by manual massage or light mechanical vibration to disperse the effusion, to promote repair and to prevent adhesions. Adhesive plaster strapping is added in such a manner that it holds the torn fibers in approximation, if the patient suffers much pain on motion. In about two weeks, work can be resumed if the tear was minute. Older cases, with the organization of effusion and a deposit of fibrous tissue within the muscles and muscle sheaths, have to be subjected to intensive physical therapy. Superficial and deep hyperemia produced by radiant light and by diathermy, respectively, followed by faradism and massage to dissipate the exudate thus created, will effect

2. Grassheim: Indirekte Muskel- und Sehnen-risse in der Unfallsmedizin, Monatschr. f. Unfallsheilk. **29**:313 (April) 1922.

3. Leusden: Ueber Muskelrisse, Med. Klin. **19**:353 (Oct. 14) 1923.

a cure in about four to five weeks. Pain on motion is an indication of the necessity for immobilization by means of adhesive plaster or a pelvic belt with a spring back extension.

2. A tear in the calf muscles may happen in mountain climbing, jumping, running or in a brisk turning of the body with the feet stable on the ground, or in walking up grade and stumbling against an unevenness of the ground. The patient feels a sudden sharp pain in the calf as if he were hit by a stone. He is hardly able to walk even with aid and may have to be carried off the place of accident. Excluding fractures and ruptures of tendon or muscle, the diagnosis is not difficult, but embolism or local thrombosis may have to be considered as possibilities. In embolism, the onset is also sudden, with pain and loss of muscle power, but in addition, there will be blanching of the skin, the absence of pulsation in the posterior tibial artery, palpable behind the inner malleolus, and cooling of the extremity. Embolism is substantiated by the presence of signs of cardiac disease or aneurysm. Local thrombosis develops slowly and has as premonitory symptoms cramps in the muscles and sensory disturbance. Pulsation is absent, and there is general evidence of arteriosclerosis. In venous thrombosis, the extremity is cyanosed and swollen, and sensations are not impaired. The thrombosed vein may be palpable, is tender, and varicose veins are in evidence. Arterial pulsation can be detected. If the laceration involves the tendinous portion of the calf, a roentgenogram will confirm the diagnosis and reveal the site and extent of the laceration. The treatment should aim to restore the function at the earliest possible date. In mild cases, the patient should at once attempt to walk with the aid of a cane. This will prevent the formation of adhesions which are liable to cause pain later from stretching of the fibrous tissue when activity is resumed after enforced rest. Walking is painful at first, but in two or three days pain will recede; normal walking will return in about two weeks. In severer cases, it may be necessary to apply a snugly fitting elastic bandage and to subject the leg to the physical therapy outlined above. The patient should be advised to walk more and more each day and to discard the bandage as soon as possible. Recovery is to be expected in about four weeks, although mild pain may persist for several months.

3. A tear in the quadriceps muscle belly comes about in one of two ways: the person may be threatened with a fall, holds himself erect with difficulty, and suddenly experiences a sharp pain in the thigh, accompanied, perhaps, by a crackling sensation, after which he falls to the ground; or, he may fall with the knee extremely bent, while the body, reclining backward, acts as a counterpulling force upon the extensors. Thus are produced not only tears in the muscle, but rupture of the quadriceps muscle, of its tendon, the ligamentum patella, as well as fracture of the patella. The diagnosis of tear is based on the findings of

local tenderness, ecchymosis, a depression in the muscle at the site of tear, on the diminished power of extending the leg and an increase of pain on flexing the knee, and on the exclusion of any other, more grave, possibility. The loss of extension of the knee is in proportion to the degree of discontinuity of muscle fibers. In severe tears, the proximal fibers contract and form a swelling; contraction against resistance makes this swelling larger and harder. The swelling may be mistaken for a muscle hernia, but the latter is prominent only when the muscle is at rest; when it is contracted, the mass diminishes in size and disappears with contraction under resistance.

The treatment aims not only to restore function but also to guard against muscle atrophy. This being essential in all muscle injuries, it is particularly essential in affections of the quadriceps which, of all muscles in the body, is the quickest to waste. Physiotherapy should therefore be administered at the earliest date. Walking with a limited range of flexion should be encouraged; violent exercises, interdicted. Strapping with adhesive to hold the torn fibers in approximation and to limit the amount of flexion should be practiced. Failure after four weeks of conservative treatment may serve as an indication for the suturing of the torn muscle, which is to be followed immediately by physiotherapy.

4. A tear in the adductors of the leg, the rider's thigh, is brought about in the manner previously described. The onset is sudden with accompanying pain and tenderness; the symptoms do not differ materially from those present with tears in other muscles of large volume. Differentiated from muscle hernia or muscle rupture, the diagnosis is established. The treatment is conservative, with physical modalities and immediate exercise within the limit of pain. Normal function returns in about four weeks, although the muscle may continue to ache somewhat longer.

5. Tears in the biceps of the arm are comparatively frequent, but practically all present a rupture of the tendons of the long or the short heads of the biceps, while the muscle substance remains uninvolved. These cases therefore do not come within the scope of this paper.

Without reporting all cases which came under my observation, three cases of uncommon etiology will be mentioned briefly.

### REPORT OF CASES

**Case 1.—A truck driver, aged 42, jumped off his truck and suddenly felt sharp pain in the anterior surface of the right leg. When he jumped, he landed on the tips of the outer three toes with the foot remaining in supination. The pain in the leg was so severe on plantar flexion that he could not manipulate the foot brake, and he walked with a noticeable limp. The next day the leg presented the following symptoms: swelling, ecchymosis and tenderness at about the middle of the anterior tibial region; the foot was in mild equinus,**

about 10 degrees plantar flexion, and could only be slowly flexed to a right angle. There was a groove in the muscle substance of the tibialis anticus and a small prominence above it. On voluntary contraction, the prominence became harder and somewhat larger. The roentgen-ray examination was negative as far as bone and soft tissue changes were concerned.

*Diagnosis.*—The case was diagnosed as a tear in the tibialis anticus muscles.

*Treatment.*—The foot was strapped into a right angle position and physiotherapy instituted. Walking with the limited amount of plantar flexion was advised. The patient recovered in three weeks after nine treatments.

CASE 2.—A woman, aged 29, had been under the care of a chiropractor for alleged rheumatism of the left leg. The usual spinal "thrusts" were administered daily. In the course of "thrusting" and manipulating the shoulders, she felt a sudden sharp pain near the vertebral border of the left scapula at about the upper third. The treatment was stopped. The next day I found the patient holding the left shoulder backwards and complaining of pain between the border of the scapula and the spine. Any attempt to bring the shoulder forward caused severe pain at about the junction of the upper and lower two thirds of the scapular border, a finger's breadth from the bone. There was sharp tenderness on pressure over this area. The contraction of the rhomboid muscles was weak and painful. No sensory or motor disturbances in the arm or forearm were present. The roentgen ray did not reveal anything pathologic.

*Diagnosis.*—The case was diagnosed as tear in the rhomboid minor muscle.

*Treatment.*—Strapping of the shoulder backward and limiting the forward motion gave instant relief. The patient received five light exposures and massage treatments. In sixteen days, she was free from pain, and was discharged as cured.

CASE 3.—A man, aged 37, while pushing a truck loaded with fruit up an incline of about fifteen degrees, had suddenly found it necessary to push harder over a one-half inch board which protruded on the surface. Experiencing a sharp pain in the back of his left leg just below the popliteal space, he stopped work and went to bed with an alleged injury to his knee. The knee did not swell but the back of it was painful when the leg was in extension. Examination, three weeks after the injury, revealed the presence of tenderness in the back of the upper end of the leg. Pain was elicited when the knee was extended and the foot dorsally flexed or when straight leg raising was attempted. The pain was always in the same place, at about the outer head of the gastrocnemius muscle. The roentgen-ray examination was negative, and all possibilities of knee joint injury were excluded.

*Diagnosis.*—The case was diagnosed as tear in the gastrocnemius muscle.

*Treatment.*—The patient was instructed to use the leg moderately and to have the heel raised one-half inch, as well as to apply an elastic bandage to the leg. The result of the treatment could not be verified because the patient never reported again.

## SUMMARY

1. The occurrence of muscle tears is more frequent than their recognition, and report in literature.

2. The characteristic physical signs at the site of the injury may not all be present. The diagnosis should therefore be established from the history with full understanding of the manner of force and mechanism

responsible for the production of the injury. The clinical findings must be the logical sequels of the history of the injury and the patient's complaints.

3. Treatment should be conservative with physical measures and early motion, which may be only partially limited if the pain on motion is excessive. Operative procedures should be considered only after thorough conservative attempts have failed.

# MYOSITIS OSSIFICANS IN THE ARMY FOLLOWING HORSEBACK INJURIES TO THE THIGH

ALBERT BOWEN, M.D.
Captain, Medical Corps, U. S. Army

BALTIMORE

A case of myositis ossificans in an army officer following a trauma received while riding led me to investigate the prevalence of this condition. I was interested in determining whether this condition, or the so-called riders' bone, is common in our cavalry.

Knowing his interest in bone tumors, I referred my patient to Dr. Bloodgood, who called my attention to the case of another army officer and most kindly gave me access to his large compilation of cases of bone tumors. After I had reviewed this large group of cases, had searched the literature, and sent a questionnaire to the surgeons of all our cavalry regiments, I came to the conclusion that these two cases were sufficiently unusual to be worth reporting.

## REPORT OF CASES

CASE 1.—W., aged 40, a captain in the ordnance department, was somewhat bowlegged, and both knees had been enlarged since childhood. He had ridden horseback since he was 7 years old, but had been riding considerably more in recent years. In 1905, he sprained his right knee and had an effusion for some time, but no permanent disability. He had a clean appendectomy performed in 1913, and there was no ossification in this scar. An examination made in February, 1923, disclosed no defects except a moderate bowing of the femurs and the tibias, with marked enlargement of the bones of both knees and an apparent outward displacement of the joints.

A roentgen-ray examination made by Major Pillsbury at the Army Medical School revealed the fact that both knees showed marked production of bone at the joint margins and at the upper and lower margins of the patella. This was much more marked on the right side. In addition, the inner tuberosity of the right knee was flattened, especially at its inner side. The inner condyle of the femur was elongated on that side, so that the joint surface on that side was oblique. The lipping on this inner side was considerable. About the margins of this right knee were three calcified bodies, apparently loose, each about 1 cm. in diameter. These were located at the time of examination, (1) opposite the intercondylar notch posteriorly, (2) back of the outer condyle, and (3) above and behind the outer condyle. The impression of the examiner was that an old injury or a faulty development in early life, which had displaced the inner aspect of the inner tuberosity of the tibia downward, had resulted in an unequal bearing surface of the right knee, with consequent chronic joint changes. A roentgen-ray examination of the hip joints was negative.

Sept. 25, 1923, when the patient was riding cross country, the stirrup came off as the horse was jumping a fence, and the patient came down with great force, striking his left thigh and groin on the saddle. He suffered great pain, but was able to ride for two hours afterward. He remained quiet for the next four days, not suffering greatly, and was able to ride for an hour on the fourth

day. The next day, he reported to the army physician in considerable pain, stating that both pain and swelling had increased after his ride the day before. An examination revealed marked ecchymosis extending from above Poupart's ligament to the left knee. The penis and scrotum also were dark and swollen. There was what appeared to be a hematoma, with marked tenderness in the region of the insertion of the adductor muscles to the pubic bone. There was no limitation of motion nor any indication that the muscle was torn from its insertion. The patient was allowed to continue his office work, but was advised to restrict the use of the leg. In about two weeks, the ecchymosis cleared up, and he suffered no disability.

About five weeks after the accident, he noticed a lump at the site of injury, and, November 19, he reported because of this condition, which caused no pain and only a slight restriction when he bent down. Examination showed the presence of a visible tumor in the femoral triangle. On palpation, there was a hard, firm mass extending from the pubis to Poupart's ligament and about 8 cm. down the adductor longus. The tumor measured about 8 by 4 cm. It felt like bone, was like bone in consistency, and was slightly movable. There was considerable induration around it, but it was not tender or painful. A diagnosis of myositis ossificans traumatica was made, and the patient was referred to Dr. J. C. Bloodgood because of his knowledge of bone tumors.

Dr. Bloodgood's diagnosis was ossifying myositis in the region of the obturator foramen and pubic bone, on the left side.

According to a roentgen-ray examination made by Dr. Max Kahn, Baltimore, November 19 and December 17, the heart and lungs were normal except for some calcified glands in the region of the right and left hila. There was new bone formation in the region of the left obturator foramen, apparently arising from the ramus of the left ischium about the junction with the ramus of the pubis. It appeared to extend into the soft structures and the region of the buttock. The shadow extended well down the adductor region parallel approximately with the femur. The hip joints were negative. The mass was not as dense as the adjacent bone, and was not of homogeneous consistency. Structural lines were not made out. The impression was that the condition was myositis ossificans traumatica. The Wassermann reaction was negative.

The mass had tended to get smaller and harder. It had caused no especial inconvenience, and had not kept him from riding. An examination, Feb. 14, 1924, revealed the fact that the tumor was smaller, harder and more discrete. The edges were now palpable, and were irregular. One could almost feel around it. All soft part induration had disappeared; it could be palpated up to its attachment to the pubic bone, and was slightly movable. Under the circumstances, operation or other interference was not considered.

Case 2.—P., aged 37, a major in the cavalry, who had accidentaly incurred several fractures during his youth, and in previous horseback accidents, and had made good recoveries from them all, in 1918, received a perforating gunshot wound of the upper left thigh; the point of exit was below the ischium in the left buttock. Recovery from this wound was rapid and complete, and he returned to the front, with apparently no disability as a result of this wound.

The patient rode as a member of the army riding team, and in November, 1920, he suffered a severe strain on his left thigh when he put his horse over a jump and the animal swerved. Immediately, a severe pain was felt in the region of the scrotum, and the patient was unable to move his legs. He was lifted from his horse and carried to the hospital. Four days later, he forced himself to ride in a horse show, and suffered greatly then and afterward. The

whole inner side of his thigh turned black and blue, a condition which gradually cleared up. He was forced to use crutches for four weeks, but the discomfort that he experienced gradually disappeared.

In the summer of 1921, he first noticed a lump just at the left of the base of the scrotum. This lump was not painful, but its size surprised him. It was firm and immovable. He had noticed no change since that time, and had taken long, hard rides and played much polo without any inconvenience and with little discomfort.

A civilian physician called his attention to the possibility of sarcoma, and he had been under observation since that time. The late Dr. A. D. Parce wrote: "We are inclined to believe that it is a type of exostosis or rider's bone, rather common among cavalrymen, and nonmalignant."

An army roentgen-ray examination, in January, 1922, revealed a growth of bone extending from the pubic bone to the obturator foramen. It was smooth, and showed no evidence of bone destruction. Another roentgen-ray examination, in February, 1923, showed that the bony growth was apparently filling the obturator foramen, but that there was no bone destruction.

According to the roentgen-ray examination of April 4, 1923, the obturator foramen was filled with a new growth of bone, and there seemed to be evidence of a new growth of bone above the lesser trochanter along the neck. By April 29, 1923, the obturator foramen was filled, and there was nothing on the trochanter or neck.

Dr. Bloodgood, to whom this case was referred by Major Parce in February, 1923, said at that time: "If we could be certain from these roentgen-ray examinations that there was any definite new growth on the lesser trochanter or the neck, this would exclude malignancy, because there would be multiple lesions. When we study the growth of bone from only the pubic bone and compare the pictures taken in January, 1922, and those taken a year later we can see no evidence of new growth of bone, no bone destruction, no soft part tumor shadow. In the roentgenograms, it is a benign lesion." Dr. Bloodgood's diagnosis was periostitis ossificans traumatica.

June 6, two plates were made by Dr. Max Kahn of Baltimore. The posterior-anterior plate showed a bony mass arising from the ramus of the pubis and extending down in front of the obturator foramen, but not occluding it. The mass was made out very distinctly as being separate from the pubic bone, with a clear line along the periosteum. It was denser than the pubis, but not of uniform consistency. Irregular lines of lesser density extended through it in several directions. The mass stood out more clearly than in any of the previous roentgenograms. It was approximately 5 by 5 cm. and pyramidal in form. There was an area of denser tissue from the lesser trochanter, parallel with the neck of the femur, probably from a soft part structure. The anterior-posterior plate showed a shadow of irregular but bony density extending over the obturator foramen. No bony growth was seen on the lesser trochanter or neck. The impression conveyed was that the condition was myositis or periostitis ossificans.

A similar condition resulting from a different trauma is given in the following case, reported by Carleton.[1]

A man, aged 45, seen in September, 1921, had a bony tumor about 4 inches (10 cm.) long and 1 inch (2.5 cm.) in diameter, projecting downward and

1. Carleton, Dudley: Boston M. & S. J. **188**:387 (March 22) 1923.

outward, in the right groin. The tumor had a flexible attachment to the pubic bone, and probably was of the adductor longus muscle; it caused no pain or inconvenience of any kind. Twelve years before, while he was sitting on the floor, tailor-fashion, he had sprung to his feet, without using his hands, and sustained an injury in the right groin. At first, he was confined in bed by a large ecchymosed tumor in the groin, which slowly decreased in size and became bony in consistency. The tumor reached its present state of growth four months after the injury, and from that time had neither increased nor diminished. A roentgenogram revealed a bony tumor, extending downward and outward from the pubis.

Bloodgood [2] cites a case reported by Graf of a man, aged 57, who received a severe trauma on the inner side of the right thigh. The patient was knocked down, but was able to get up and continue work. He limped for three months, suffering increasing pain and disability at the hip joint. Four and one-half months after the injury, he was confined to bed on account of the increase of symptoms.

According to an examination made eight months after the injury, the right thigh was flexed 45 degrees, adducted 35 degrees, and rotated out. No motion was possible in the hip joint; there was no shortening nor any sign of hip joint disease. Extending between the femur and the pelvis in the adductor muscles, there was a hard, bony mass. The skin was normal. The patient was able to walk painfully with the assistance of two canes. Under ether, forced adduction separated the bony, new growth from the shaft of the femur and permitted correction of the deformity. This relieved the patient, who would consent to no further operation. The roentgen-ray shadow of the mass was of the same density as that of the femur, and the tumor was of eight months' duration.

## PREVALENCE

Many cases of myositis ossificans have been reported in the literature, and, although the adductor region of the thigh is one of the places where it tends to occur, the cases in which it occurs in this region are not numerous. Probably because of the greater exposure to injury, young males are more prone to this affection.

The following summary of the number of cases reported illustrates the comparative rarity of myositis ossificans in the adductor longus:

Binnie [3] compiled eighty-six cases in 1903, three of which were of the adductor longus. Makins [4] refers to Schultz, who compiled 233 cases in the German army, of which all but three were of the quadriceps or brachialis anticus. Painter [5] refers to Sir Robert Jones' compilation of 339 cases in 1912, the vast majority of which occurred in from early to middle adult life, predominantly in males—only thirteen being in females. The commonest muscles involved were the brachialis anticus,

2. Bloodgood, J. C.: Progr. Med., December, 1902; ibid. December, 1913; J. Radiol. **3**:310 (Aug.) 1922.

3. Binnie, J. F.: Ann. Surg. **38**:423, 1903.

4. Makins: Proc. Roy. Soc. Med. **4**:134, 1910-1911.

5. Painter, C. F.: A Consideration of the Etiologic Factors in Myositis Ossificans Traumatica, Boston M. & S. J. **185**:45 (July 14) 1921.

quadriceps femoris, adductor longus, and the biceps of the upper arm, in the order named. The exact number occurring in the adductor longus was not stated. Coley [6] refers to Straus's compilation of 127 cases of traumatic ossifying myositis only thirteen of which were of the adductors of the thigh.

A brief questionnaire was sent to the surgeon of each of the cavalry regiments of the United States army stationed in the United States, asking for case reports of riders' bone or myositis of the thigh following horseback injuries. A majority of the surgeons replied, and it is assumed that the remainder had nothing to report, since all of those heard from agreed that, from the records consulted and according to their personal experience, there had been no cases of riders' bone or of myositis ossificans in their regiments. Dr. D. W. McEnery, however, referred to the case of P. (Case 2), who had been at Fort Riley. It would seem, therefore, that one is justified in concluding that the condition is uncommon among our mounted troops.

## CLASSIFICATION

Most authors agree in dividing myositis ossificans into three distinct groups as follows: (1) Myositis ossificans progressiva; (2) a localized form due to chronic irritation from relatively slight traumas, and (3) myositis ossificans traumatica conscripta.

1. Myositis ossificans progressiva is a constitutional disease which begins usually in the young, and is widespread throughout the skeletal muscles.

2. In the class of the localized form of ossificans due to chronic irritation from a series of relatively slight traumas to some particular region are put the small masses that are sometimes found on the inner side of the thigh and leg in jockeys. Binnie [3] describes riders' bone as found in the region of the adductors of the thigh, cavalryman's bone as an osseous plate where the saber hits the outer side of the thigh, and soldier's bone as the condition found where the rifle rubs the shoulder.

Dr. A. N. Stark, in a personal communication, told me of several cases of cowboys in Texas, in which there were small deposits of bone in the thighs which he had removed during his service there, but he did not recall any cases seen in cavalrymen.

3. Myositis ossificans traumatica conscripta is produced by a single trauma. It is this group that we wish to discuss and to which my case belongs.

The term is not satisfactory, for many deny that the process is inflammatory and many names have been suggested. Morley [7] prefers

6. Coley, W. B.: Ann. Surg. **57**:305 (March) 1913.
7. Morley: Brit M. J. **1**:1475 (Dec. 6) 1913.

the term "traumatic intramuscular ossification." Stone [8] calls it "traumatic ossification." Fay [9] suggests "parosteal callus." Oliver [10] is of the opinion that the so-called parosteal callus, exuberant callus, and myositis ossificans following a single trauma are genetically the same.

It would also seem that ossifying traumatic periostitis and traumatic exostoses belong to the same group of lesions, although they do not invade muscle tissue to the same extent. Although the history and findings of Cases 1 and 2 are almost identical, Bloodgood calls Case 1 myositis ossificans and Case 2 periostitis ossificans traumatica.

## ETIOLOGY

The etiology of myositis ossificans is its most obscure factor, and it is on this point that the greatest amount of discussion hinges in the literature. Painter [5] has reviewed the literature very carefully, and reports one recent case involving the quadriceps femoris.

Binnie [3] reviews the literature up to 1903, and discusses the etiology. It is not necessary to review all the theories that he found. Most investigators have implicated the periosteum as the source of the new bony growth. Many ways have been suggested, and I quote the two most plausible. Berndt believes that a severe blow is inflicted. The muscle affected is crushed into a pulp. There is injury to a limited and adjacent area of periosteum. The cells of the injured periosteum proliferate, escape into the pulpy area of muscle, and there develop. A mass of bone is formed which may have only a loose connection with the periosteum or may became entirely detached. Lyot's theory is:

> A muscle is in action, receives a blow; some of its fibers tear themselves from their bony origin and retracting into the mass of muscle still connected with the bone, carry with them particles of periosteum. These particles of periosteum grow in their new environment, are true bone grafts and can form intramuscular osteomata, unconnected with, though originating from the periosteum.

Ziegler, also quoted by Binnie, assumes a congenital diathesis of the connective tissue of the muscles, fascia, tendons and ligaments, in that they become endowed with powers usually belonging only to the periosteum. In the case of the progressive type of myositis ossificans, such a theory of metaplasia seems the most acceptable.

Sir Robert Jones, quoted by Painter,[5] is of the opinion that the majority of his 339 cases were due to osteogenesis of avulsed periosteum which, when it escaped into the muscles enveloping the bone from which

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8. Stone, D.: Arch. Radiol. & Electroth. **13**:225 (Jan.) 1919.
9. Fay, O. J.: Surg., Gynec. & Obst. **19**:174, 1914.
10. Oliver, Paul: Myositis Ossificans Following a Single Trauma, J. A. M. A. **63**:1453 (Oct. 24) 1914.

the periosteum had been torn off, continued to grow. True bone is thus developed which cannot be distinguished from callus.

Finney,[11] in 1909, reporting six cases, thought that many cases came from tangental injuries by kicks or football bruises, mostly on the front of the thigh. When a muscle is taut and sustains a violent trauma from the side, muscle fibers are easily torn from the bone and would carry fragments of periosteum along with them. He believed that it is rare in fractures or crushing injuries.

Stone [8] calls attention to the fact that traumatic ossification occurs in muscles in which there is practically no tendinous tissue interposed between the muscle and the bone itself. He also objects to the term myositis in describing these tumors.

Fay [9] reviews the subject from the first descriptions by Copping, in 1740, and Freke, in 1741. He objects to the term myositis, and discusses the origin and nature of the new formed bone. He favors the term "parosteal callus," but says that, until it has become possible to produce these parosteal bone masses experimentally and constantly, any explanation must be more or less hypothetic.

Morley [7] considers the process to be somewhat as follows:

> A severe blunt injury at once subcutaneously strips off and destroys the periosteum and crushes the muscles in contact with the bone. Bleeding occurs from the surface of the denuded bone and with the blood osteoblasts in a free and possibly ameboid condition escape into the pulped muscle tissue and blood and there produce a growth of bone. Healthy muscles left in contact with denuded bone form protective fibrous adhesions from their interstitial connective tissue and take on the limiting function of the periosteum. But severely contused muscle tissue mixed with blood provides a favorable pabulum for osteoblasts.

From the careful pathologic study that he made, Binnie favors Ziegler's assumption of a definite diathesis by which a metaplasia of one form of connective tissue to another may take place, the bone tumor being the result of proliferation and metamorphosis of the interstitial connective tissue of the muscle.

Davis and Hunnicut [12] made extensive animal experiments and studies on the growth of periosteum and concluded that: (1) Periosteal transplants, in the majority of cases, do not produce bone; (2) pedunculated flaps of periosteum produce no bone, except for the pedicle connecting them with the bone, and (3) free periosteal flaps and pedunculated flaps with bone shavings attached produce bone in every instance. These experiments support Morley's contention that the periosteum is a limiting membrane without osteogenetic powers in itself.

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11. Finney, J. M. T.: South. M. J. **3**:36, 1910.
12. Davis and Hunnicut: Bull. Johns Hopkins Hosp. **23**, March, 1915.

Lewis [13] reports two cases of his own and eleven from the literature in which myositis ossificans occurred in operation scars in the abdominal wall in which the possibility of periosteal transplant was out of the question. He expresses the belief that this class of cases is due to metaplasia.

Another group of cases following posterior dislocation of the elbow, in which new bone formed in the brachialis anticus, he believes to be due to periosteal stripping and callus formation.

Painter concludes that there is not sufficient evidence at hand to prove the case for any one of the many theories advanced as causative in the production of traumatic myositis ossificans. He favors the idea of a diathesis working somewhat as follows: Persons in the first group have a tendency to bony metamorphosis and develop the progressive type of the disease early in life; those with a lesser tendency develop circumscribed bony tumors after repeated slight traumas, while those in the third group, with a less dominant diathesis, require a very severe trauma to produce bony change in muscle.

In the case of W. (Case 1), there was apparently a diathesis which had already produced an overgrowth of bone in the knees. Then came a severe trauma, probably tearing off some periosteum with underlying osteoblasts, hemorrhage, and pulpifying of muscle, into which the osteoblasts were planted, and, on account of the bony diathesis, these proceeded to form a bony tumor in the muscle. There are so many similar injuries in which ossification does not occur that one is almost forced to the conclusion that there is some determining factor in addition to the trauma.

### PATHOLOGY

The gross pathology is well described by Morley,[7] who operated on a mass in a thigh, three weeks after injury, which was reported as sarcoma from the roentgen-ray examination. He found a large hematoma under and in the muscles, with the femur covered by a layer of new bone, averaging 2 cm. in thickness; from it, blunt spikes and bars projected irregularly into the muscle and cavity of the hematoma. It presented the appearance of a 3 weeks old callus. The mass with the periosteum was removed with a gouge, and, within three weeks, a definite recurrence, much larger than the original growth, took place.

Concluding that the cause of the recurrence was the destruction of the periosteum and the loss of its function as a limiting membrane for the osteoblasts, Morley decided to remove the recurrent bone and to transplant a piece of iliotibial band over the denuded femur to prevent adhesions and recurrence. This was entirely successful.

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13. Lewis, Dean: Myositis Ossificans, J. A. M. A. **80**:1281 (May 5) 1923.

He reproduced the condition experimentally in animals by reproducing the same mechanical conditions, denuding the femur of periosteum and crushing the overlying muscle. He also believes that he showed that osteoblasts and chondroblasts are the same cells existing under different conditions of nutrition.

Sir Robert Jones, quoted by Coley,[6] says:

> The bone is generally of the cancellous type, and at the edge of the bone the muscle seems to have been sprinkled with numerous small foci, around which the bony matter has been deposited. Generally, the center of these foci contains a small branched cell (osteoblasts). The bony matter is deposited along the muscle fibers and the striation of the muscles may still be visible.

Ewing, quoted by Coley,[6] in describing a section, states:

> It is composed of numerous trabeculae of bone, round, elongated, branching, and anastomosing as in callus formation. The masses are usually well calcified but some are deficient in ossification, often surrounded by numerous large osteoblasts which are evidently in the process of bone formation.

Some hold that the condition is akin to sarcoma; this is borne out by the following cases: Coley[6] gives a very complete history, with numerous roentgen-ray plates, showing myositis ossificans traumatica of the femur in a female, in whom the condition became sarcoma six years later. Three years after injury, a section was removed for the purpose of making the diagnosis, which was myositis ossificans. Nearly three years after this, the mass enlarged, and Coley removed it from the muscles and bony attachment. Dr. Ewing found giant cells in one area of what he considered a mass of myositis ossificans. Within three months, a large, soft tumor, evidently sarcoma, had developed in the region of the wound. A section was made and the case was pronounced giant cell sarcoma by Dr. Ewing. Amputation was performed, but the patient died, about eight months after the operation from metastases of the spine.

Bloodgood regards this case as a very malignant, mixed cell sarcoma containing giant cells, rather than a malignant giant cell tumor.

DaCosta[14] tells of operating in a case of myositis ossificans traumatica, and removing an ossified area in the thigh muscle. The pathologic report was myositis ossificans. The growth quickly recurred but showed little ossification. The pathologic report then was spindle cell sarcoma. This patient and another patient of DaCosta's reacted to Coley's fluid.

Keinboeck,[15] in a careful roentgen-ray study of this condition, states that in the third or fourth week after injury the mass first becomes apparent as a faint indistinct shadow, separated from the diaphysis by a

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14. DaCosta, J. C.: Modern Surgery, Philadelphia, W. B. Saunders Company, p. 713.

15. Keinboeck: Wien. klin. Rundschau **17**:845, 1903

zone of light. This bright line seems to be very constant except where the mass straddles the bone. The shadow is usually long and slender, running parallel to the long axis of the bone. The shadow is by no means homogeneous, but consists of varying densities representing different stages of bone formation. For several weeks, the picture continues to change; the shadow grows darker and more homogeneous; it shows figuration, stripes and spots, the latter being in turn shaded by the formation of lamellae, which are in part arranged parallel to each other. After from four to six weeks, the shadow volume corresponds closely to the palpable mass, and it is probable that no further appreciable changes occur. A broad zone of light between mass and bone signifies that it is not attached. The narrow zones of light are not conclusive.

Lewis [13] also gives a roentgen-ray picture of myositis ossificans: the roentgen-ray shadows are at first indistinct and diffused but gradually become smaller and denser. Islands of bone are seen in the mass. The shadows are not homogeneous. Trabeculae may run with the muscle fibers. The tumor is slow in reaching its final form. Often the shadows do not appear to be connected with the bone. At an early stage, there may be only a roughening of the bone resembling accentuated muscle attachment. There is no medullary change or destruction of the cortex. The shadow at either extremity of the mass is separated from, rather than continuous with, the periosteum, and is parallel with the shaft of the bone. It shows a dotted veil appearance, and there is no periosteal stripping.

### DIAGNOSIS

Common conditions from which a diagnosis must be made are: contusions, hematoma in the muscles, myositis, interstitial syphilitic processes, gout, muscle tumors, osteomyelitis, muscle callus, exostoses, traumatic periostitis, infectious ossifying periostitis, and sarcomas.

A Wassermann test should be done in every case.

As in the case of P. (Case 2), the same injury and course may produce either exostosis or myositis. The exostosis has a more intimate connection with the bone, and there is no actual change of muscle tissue into bone.

In periostitis, the new bone is formed in the periosteum and may invade surrounding muscle. Bloodgood [2] describes several cases of infectious ossifying periostitis, in which there is a new growth of bone which is always intimately connected with the periosteum. There is no history of trauma, and careful search reveals some focus of infection. The roentgen-ray examination shows the tumor to be continuous with and part of the periosteum, while ossifying myositis always shows a clear line of separation from the shaft of the bone or a marked clear zone of separation.

Of all the conditions from which ossifying myositis must be distinguished, the one of most vital and paramount importance to both patient and surgeon is sarcoma. That this diagnosis is not easy, and that many limbs have been needlessly sacrificed with resulting "sarcoma cures," is evident from a perusal of the literature. Three of the patients in the six cases reported by Finney came with the diagnosis of sarcoma. Bull [16] reported four cases of myositis developing from three weeks to several months after trauma, with hematoma, and in three of the four cases sarcoma had been diagnosed.

P. (Case 2) was sent to Dr. Bloodgood because of the possibility of sarcoma.

In the article to which previous reference has been made, Coley differentiates the diagnosis from that of sarcoma in the bone. The age of the patients and the fact that the tumor develops shortly after injury furnish a history almost identical with that often observed in patients with sarcoma. Even the roentgenogram may appear similar. Sarcomas are more likely to develop near the epiphysis; myositis ossificans usually comes from the shaft. Sarcomas are rarely uniformly hard; some areas are soft and yield to pressure. Sarcomas increase steadily in size while myositis ossificans tends to shrink after a period of growth, usually after two or three months. The pain usually comes early in myositis and lessens in severity, while in sarcoma it comes later and tends to ever increasing acuteness.

The roentgen-ray examination will usually furnish a means of diagnosis. The cortex is not invaded in myositis, and shows no change in appearance. In sarcoma, the cortex is early invaded. In myositis, the junction of the tumor and bone is sharply defined and the periosteal line can be traced, while in sarcoma it is less distinct, except in the very early stages.

Lewis [13] makes the following points in the differentiation from periosteal sarcoma: the cortex is usually eroded and irregular; there are stalactitic processes, usually at right angles to the shaft of the femur; there is a fusiform extension of both ends of the shadow onto the shaft, while the shadow of myositis ossificans is laid down in lines parallel to the shaft, and crossed by others more or less at right angles.

Coley [6] thinks that the periosteal sarcoma is less clearly defined by the roentgen ray and shows frequent indentations of the tumor mass on the side toward the bone from which it is derived, while in myositis ossificans there is a clearly marked line of separation between the tumor formation and the shaft. Reference has been made to one of Coley's cases that changed from myositis to sarcoma.

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16. Bull, P.: Norsk Mag. f. Lægevidensk. **83**:992 (Dec.) 1922.

Most surgeons now agree that, in cases of difficulty of diagnosis from sarcoma, an exploratory operation or an excision for purposes of diagnosis should be made before amputation is considered. In fact, the mortality from periosteal sarcoma is nearly 100 per cent., regardless of the treatment; thus, exploration does not increase the danger, and amputation does not offer a cure. Dr. Bloodgood's present procedure is not to cut into them at all, but to make the diagnosis from roentgenograms and, in cases in which sarcoma is believed to be present, to treat by deep roentgen-ray therapy.

## TREATMENT

In the treatment of myositis ossificans traumatica, every sort of measure has been tried. Several surgeons have reported very carefully the results of early, middle and late operations checked up with roentgen-ray and pathologic examinations. Their wisdom may be simply summarized: "If it's causing no trouble, leave it alone."

Painter [5] describes a case in a thigh in which he removed the growth early, with a recurrence resulting in a month. However, it did not recur after the second removal, and he concluded that he operated too soon.

Morley [7] also found that a simple, early excision was usually followed by recurrence. He also found that conservative treatment in some cases was the cause of long or permanent disability. He found that excision, combined with grafting of the deep fascia on the denuded bone surface, gives the best prospect of rapid and complete recovery, and it is urged as the treatment for all cases not complicated by ossifying periarthritis.

Finney [11] warns against too early operation because of the tendency to recurrence, and because there is likelihood of spontaneous shrinkage and degeneration.

Godlee [17] says, "The operation is inflicting another traumatism upon a part which for some reason has shown a special tendency to the development of bone and it cannot therefore be surprising if renewed activity of the process should follow."

While some of the continental surgeons favor early excision, nearly all others are in agreement as to conservative treatment, favoring letting the growth alone unless it interferes with function, and then operating only after osteogenesis has subsided and the process is stationary.

## SUMMARY

1. Myositis ossificans and riders' bone following horseback injuries are not common among our mounted troops.

2. The etiology is not absolutely determined. It is probable that both a diathesis and an exciting cause are necessary to its production.

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17. Godlee, R. I.: Tr. Roy. Soc. Med., 1911.

3. The principal problem in diagnosis is to rule out sarcoma and not to treat this benign lesion as malignant. A good roentgenogram will usually enable the diagnosis to be made. When in doubt, an exploratory excision for diagnosis is permissible.

4. Conservative treatment, without operation unless function is interfered with and then only after all growth has ceased, is the rule.

## DISCUSSION

Dr. Joseph Colt Bloodgood, Baltimore: This affords another opportunity to try to make clear the differential diagnosis between benign and malignant ossifying lesions.

First, I must take the opportunity to thank Dr. Bowen for reporting two very typical cases in an unusual position, and for his very concise and unusually clear discussion of the differential diagnosis and the more important points in the literature. If one reads this article and the American references alone, it will not be necessary to go further to obtain a good idea of ossifying myositis.

Nevertheless, in spite of our increasing experience, the chapter on the differential diagnosis between benign and malignant ossifying lesions is not yet completed.

Again and again, before and especially since the roentgen ray came into use, I have restudied all my recorded cases and the literature to get exact data for this important differential diagnosis. Nevertheless, when the real test case comes, all methods—clinical, roentgen-ray, gross and microscopic—may fail.

1. When the roentgen ray pictures a clear zone between the shaft and the dark area of bone formation in the soft parts, and this dark area is distinctly that of osseous tissue, sarcoma can be excluded.

Tumor shadows and outlines in the soft parts due to tissue not containing bone, with the rarest exceptions, can be easily distinguished from tumor shadows containing bone, but the differentiation between benign and malignant soft-part tumors, so far, seems impossible from the roentgen-ray examination.

The majority of cases of ossifying myositis belong to the first group. The roentgenogram shows a zone of bone formation in the soft parts with a clear zone between it and the neighboring bone of the skeleton. The treatment, as outlined by Dr. Bowen, is distinctly one of noninterference.

2. When the shadow in the roentgenogram of the new bone formation touches, overrides or surrounds that of a neighboring bone of the skeleton, an ossifying periostitis must be considered, and a possible ossifying periosteal sarcoma must be borne in mind.

3. In one group of the type described in Group 2, there is, for practical purposes, both an ossifying periostitis and an ossifying myositis. The size, the shape and the markings of the shadow of the new bone formation are so characteristic that the benign lesion can be recognized in the roentgenogram just as readily as in Group 1, in which there is a clear zone about the neighboring bone of the skeleton. The two cases described by Bowen in his paper belong to this group, Group 3.

4. Fortunately, this is the smaller group. The shadow of the new bone formation is chiefly that of an ossifying periostitis and not unlike an ossifying

sarcoma. But, in a few cases in the benign and particularly in the malignant form, the bone formation may be excessive and suggest an ossifying myositis.

My studies emphasize the point that an ossifying sarcoma may suggest in the roentgenogram ossifying periostitis and ossifying myositis.

I have discussed this point, with illustrations and cases, in the *Journal of Radiology* (4:119 [April] 1923).

If one can see clearly the shaft of the bone of the skeleton, and there is no bone destruction, and the surface of the cortical bone is smooth, and the marrow cavity unchanged, this evidence is against sarcoma. But, recently, I have seen roentgenograms of cases later proved to be sarcoma, in which, beneath the bone formation, the cortical bone and marrow shadow had all the appearance of the normal.

On the other hand, a new bone formation may so obstruct the view of the bone that it covers or surrounds, that it is impossible to tell whether there is bone destruction or not.

Again, in some forms of sclerosing, nonsuppurating osteomyelitis, we may have this excessive new bone formation with evidence of bone destruction in the cortical bone and a shadow of a marrow cavity not normal.

5. To repeat, there are some cases with ossifying periostitis and possible ossifying myositis in which it is impossible, in the roentgenogram, to distinguish the benign from the malignant.

### PALPATION

In one instance only, in my own experience, has an ossifying lesion surrounding the shaft of a bone felt like a benign bone formation and proved to be sarcoma. I have reported this case in the article mentioned. In this instance, the roentgenogram showed bone destruction of the cortical zone and a changed marrow shadow. I recorded that the bone tumor about the upper end of the femur below the trochanters felt like benign ossification, but the roentgenogram favored sarcoma.

With this exception, palpation has been very helpful. In the majority of cases of benign exostosis, osteoma, ossifying periostitis, ossifying myositis, sclerosing, nonsuppurating osteomyelitis and excessive callus formation from fracture, the palpation was like that in normal bone, being felt almost as clearly as the palpation of one's shin bone. In ossifying sarcoma, one, as a rule, feels on top of the bone formation a soft-part tumor, but the one exception proves that we cannot always depend on this rule. On the other hand, in ossifying myositis, especially in the recent stage, one may not be able to palpate directly the benign bone formation. Normal soft parts or soft parts changed by an inflammatory exudate may intervene.

When I first reviewed the literature in *Progressive Medicine*, December, 1902, and read Binney's classical reviews in the *Annals of Surgery* for 1903, and read over the records of my first cases of ossifying myositis and ossifying sarcoma, there was no difficulty in making the differential diagnosis by palpation alone, and, in the days before the roentgen ray, the diagnosis was usually correctly made. The same is true in the first years after the roentgenograph was introduced as an instrument of precision. This ease of differentiation by feeling or by the roentgenogram can be explained only by the fact that we saw both lesions late. Now that we see both the benign and the malignant early, we find that this small group in which both roentgen-ray examination and palpation fail to differentiate is increasing.

### CLINICAL HISTORY

It is quite true that the benign lesion of ossifying myositis and the sarcoma give different histories, but, unfortunately, this cannot be depended on. In the benign ossifying myositis, the original swelling after the trauma rarely disappears; it gradually changes into the hard bone formation; while in sarcoma there is, as a rule, no primary swelling after the trauma, and if there is one, it, as a rule, disappears. The palpable mass in sarcoma appears at varying intervals after the trauma, usually between a few weeks and a few months. In my cases, pain was more prominent in the malignant lesion.

### EXPLORATORY INCISION

Dr. Bowen has described my position correctly. The objection to exploratory incision is the difficulty of recognizing the sarcoma in the section. This statement is based on facts. A number of cases of ossifying sarcoma have been submitted to a number of trained pathologists, and their microscopic diagnoses from the section have differed. The same has been true when sections of benign ossifying lesions have been examined by a number of experienced surgical pathologists.

### CONCLUSIONS

In view of the fact that amputation rarely, if ever, has cured a sarcoma of the scapula, of the upper extremity, or of the femur above the middle third, deep roentgen-ray treatment here should always be the method of choice. There is, therefore, no necessity for the exploratory incision. When the bone lesion is situated on the lower extremity in a locality in which amputation has given a certain percentage of cures, exploratory incision should be considered. However, when the clinical picture, the roentgen-ray examination and the palpation place the ossifying lesion in the group difficult to diagnose, I am inclined not to operate.

A. When the ossifying periostitis or myositis is distinctly benign, operation is not indicated.

B. When the ossifying lesion is distinctly malignant, there is no necessity for an exploratory incision; if resection with restoration of function, with or without bone transplantation, is possible, it should be performed at once. If the tumor is below the middle third of the femur and cannot be resected, in the majority of cases, amputation should be done at once. In other localizations where resection is not feasible, roentgen-ray treatment should be depended on. At the present time, I have records of one periosteal sarcoma of the upper third of the humerus apparently well at present, three years after radium treatment by Drs. Burnam and Neill in Dr. Kelly's sanatorium. A second sarcoma, of the scapula, well almost two years since deep roentgen-ray treatment by Dr. Kahn, and a third recent periosteal sarcoma of the upper third of the shaft of the femur, have shown almost complete recession after prolonged deep roentgen-ray treatment by Dr. Kahn.

C. The benign ossifying lesions outnumber the malignant. The group easily recognized from the roentgenogram, from palpation and the history outnumbers the group in which diagnosis is difficult or impossible.

This smaller group must be restudied as our material accumulates, and my material is now ready for a second investigation and publication.

In discussing this differential diagnosis, I have considered the local condition only. One should never neglect a roentgen-ray examination of the chest, because the typical button areas mean metastasis, and positive evidence of

tuberculosis is rather against malignant disease. Throughout my entire course of study of malignant tumors, I have constantly borne in mind the possibility of malignant disease in cases in which there is clinical or roetngen-ray evidence of active tuberculosis, and the fact still remains true that the two diseases rarely occur in the same person. The opposite is true of syphilis—a positive Wassermann reaction does not exclude malignant disease, but, of course, the patient should be given the benefit of intravenous therapy, especially in bone lesions.

### THE RELATION OF TRAUMA TO BENIGN AND MALIGNANT LESIONS OF BONES AND SOFT PARTS

Myositis ossificans of the type reported by Dr. Bowen, and so well summarized by him, is never observed without a definite history of a contusion or trauma, and, as I remember the literature, the investigations and discussions of this difficult to explain ossifying myositis have lead to the study of the evidence and much philosophical and theoretical discussion on the exact relation between a single or repeated traumas and a sarcoma of the bone and of the soft parts.

A number of authorities believe, although it cannot be proved, that sarcoma never begins in healthy connective tissue, but that there must be granulation tissue first, and then some type of cells in the granulation tissue wins out in the survival, and the different types of well-known sarcomas develop. There is, first, the so-called simple sarcoma which develops from the embryonic connective-tissue cell and gives rise to the fibrospindle, spindle, round and various types of spindle and round cell sarcoma. Then there is the angiosarcoma which arises either in the endothelial or perithelial cell of the capillary of the granulation tissue.

The more I restudy the old material and get better histories in the new material, the more I feel that there is a definite relation between repeated traumas and chronic irritation and the development of carcinoma, and a single trauma with a subcutaneous wound, granulation tissue and sarcoma.

Of course, there must be other etiologic factors, or we should all die of malignant disease. This other factor is still obscure. Heredity may play a definite part.

In the skin and in the mouth, we can, by education, reduce the dangers of chronic irritation to a minimum, and perhaps in the cervix of the uterus. At the present time, we have no data that would lead to any preventive treatment for cancer of the breast. In spite of the revival of the relation of chronic poisoning from stasis, constipation and improper food, we have as yet nothing definite to suggest concerning diet as a preventive of cancer of the alimentary tract or anywhere else.

Although "safety first" and the safety mechanisms may reduce the number of cases of single trauma, we must still educate ourselves and continue our investigations of how to recognize the difference between a benign and a malignant lesion of the bone or soft parts which may follow a single trauma.

### CONTUSION OF THE THIGH

Apparently, there is no doubt that sarcoma rarely develops after an open wound or an operative wound, even when it has healed by granulation. The majority of sarcomas give a history of subcutaneous injury. Of course, we do not know the total number of subcutaneous wounds as compared with open wounds. The former may be so much greater in number that the percentage of secondary sarcomas is practically the same in both. The practical point is this:

We must bear in mind the possibility, educate the public to the facts, educate and influence them to seek examinations, and, when they do this, make a proper and thorough examination. The two most important elements are palpation and the roentgen-ray examination, and these should be repeated until all signs, pain, loss of function and palpable mass, disappear. The roentgen-ray examination should be repeated.

Now, as a matter of fact, what may follow a contusion of the thigh without fracture? Tenderness, limp, pain on motion without visible ecchymosis or palpable mass may follow. There may be the early or late appearance of extravasation of blood. Immediately, a hematoma may form; rarely, a swelling due to a lymph cyst; a ruptured muscle; a distinct nonossifying myositis; an ossifying myositis; a lipoma; a concealed fibroma or other type of benign tumor may begin to grow; the bursa in the neighborhood may begin to fill with fluid, or a sarcoma may develop. I have records in the laboratory of every one of these types, and, with the rarest exceptions, not a single patient has sought examination until the condition was fully developed. The contusion of the thigh may give rise to reaction in the bone, traumatic, syphilitic, or infectious periostitis with and without ossification; an osteomyelitis; a benign giant cell tumor or a sarcoma.

We have not had a sufficient number of cases to teach us the possibility of differential diagnosis in the very early stage. I have learned a few things from careful observation and records. After contusion to the bone, tenderness may persist months or even a year without any serious condition developing. I have been over some of these cases recently. If roentgenograms are taken at once after the contusion, we may find revealed a lesion of the bone which must have been present before the contusion, while, if the taking of the roentgenogram had been postponed, it would have been difficult or impossible to tell.

Again, if the roentgenogram showed ossification in the soft parts not connected with the bone, as far as my evidence goes, sarcoma has never developed.

From my recent experience, I am positive that we have much to learn, when patients come at once, if we will take roentgenograms at repeated intervals, palpate, record the detailed history and omit no important laboratory tests. The majority of my first patients of this type were brought to me to comply with the workmen's compensation laws.

### FRACTURE AND SARCOMA

From the very beginning of the literature, all observers record the fact that any type of tumor, benign or malignant, rarely follows a fracture, but now and then it does happen. The most common is the giant cell tumor which is not malignant. This may be somewhat explained by the fact that contusion of a bone is much more common than a fracture.

The entire subject of the relation of trauma to benign and malignant lesions of the bone and soft parts needs restudy. I have attempted it on a number of occasions, but the older records are faulty, and the good records, as yet, are in the minority.

The great lesson is: After any type of injury, the individual should seek an examination, and this examination should be recorded with the roentgen-ray examination and the other laboratory tests, and should be repeated until all local signs have disappeared.

Recently, in my laboratory in only two days, I have studied and dictated a note on at least a single case which will illustrate practically every point of fact recorded in this discussion.

# A CASE OF PRIMARY MULTIPLE ENDOTHELIOMA OF BONE

## WITH SPECIAL EMPHASIS ON ITS ROENTGENOLOGIC FEATURES *

ANATOLE KOLODNY, M.D.
CHICAGO

Thirty years have passed since Volkmann, in his epoch-making monograph on endotheliomas, pointed out that the discrepancy in the interpretation of tumors is due to the general attempts to classify them on the basis of their morphologic structure alone. He emphasized the necessity in all complex cases of tumors of studying the origin and the development of the tumor and not the cytology present and the arrangement of the cells alone. It is generally known that the differentiation of pathologically changed endothelial cells from epithelial cells is very difficult, if not impossible. There can be no question, however, that in such border line cases the study of the development of the tumor and the tracing of its origin can contribute much toward making a correct diagnosis.

It is usually considered that a primary endothelioma of the skeleton is incomparably more difficult to diagnose than nonskeletal endotheliomas. It is true that in endotheliomas of bone unfavorable conditions for a careful cytologic study are encountered. The process of fixation and decalcification is exceedingly harmful to the highly delicate tumor cells. It seems to me, however, that this same fact, namely, the location of the tumor in the bone, can be of great help to us in the clinical diagnosis. I refer to the roentgenologic study of such cases. Meanwhile, in the manifold literature of endothelioma of bone, I met with just one case in which the author called attention to the roentgen-ray picture.[1] Consequently, I propose to demonstrate the importance of this neglected side of the question by means of a rather rare case of a bone tumor, interpreted as a primary multiple endothelioma of the skeleton.

### REPORT OF CASE

Mrs. M. F., aged 48, was admitted to the Iowa State University Hospital, March, 1923, with complaints of a dull ache in the left hip, in her back between the shoulders, and in the right leg. The patient had felt well until February, 1923. Examination revealed tumors on the right leg immediately above the

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* From the University of Illinois College of Medicine.

* Presented before a meeting of the Chicago Pathological Society, Feb. 11, 1924.

1. Ewing, James: A Review and Classification of Bone Sarcomas, Arch. Surg. 4:485 (May) 1922.

external malleolus and on the left tuber ischii. The tumors felt hard and bony and the overlying soft tissues were not attached to them. The skin in these areas was normal. Roentgen-ray examination showed bony tumors on the right fibula, left tuber ischii, and on both scapulae. The blood examination was negative. Bence Jones' proteins were repeatedly absent. Early in March, a piece of tissue was removed from the fibular tumor by Dr. Beye for a histologic diagnosis. From the sections prepared by the rather crude routine method (fast decalcification, paraffin embedding) I, at that time surgical pathologist at the Iowa State University Hospital, reported that the tumor was malignant and that the diagnosis must rest between a metastatic carcinoma and multiple endothelioma. Since the condition seemed to me of great interest, I consulted Dr.

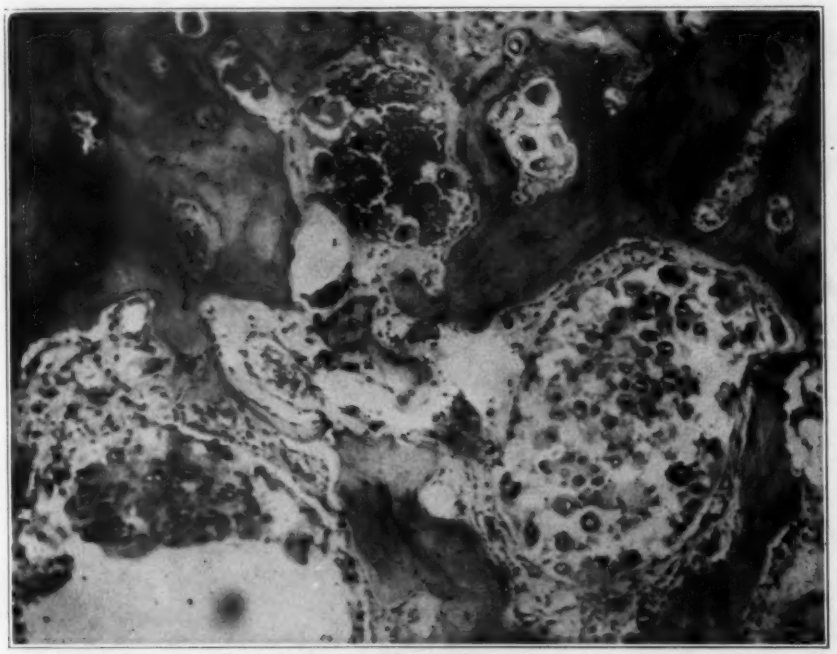

Fig. 1.—Photomicrograph, showing the proliferation and piling up of the blood vessel endothelium. Magnification: Distance, 12 inches; ocular, 4; objective, 3.

Bloodgood, to whom I sent sections of the tumor. His report coincided with mine. The patient remained in the hospital until her death in November, 1923. During her stay, she developed a tumor on the forehead. Radium and roentgen-ray therapy produced no noticeable results. The pain in the bones became so severe that in May, Frazier's operation was performed by Dr. Rowan.

The necropsy was performed by Dr. Orton. Duplicate samples of all the organs removed at necropsy were sent to me for microscopic examination while I was associated with the department of anatomy, College of Medicine of the University of Illinois. The necropsy was performed as completely and carefully as possible, Dr. Orton giving special attention to the possible occurrence of a primary tumor in the soft tissues. Microscopically, there was nothing of

interest except that the tumors of bone throughout the skeleton (fibula, scapulae, skull, ribs and pelvis) were covered with intact periosteum, and there was no infiltration of the soft tissues, which were pushed away by the tumor mass. There was a marked enlargement of the mediastinal and mesenterial lymphnodes and multiple miliary nodules scarcely visible in the lungs.

*Microscopic Examination.*—The compact portion of the involved bones seemed less dense than normal. The enlarged Haversian canals were filled with tumor cells which were very loosely arranged without any supporting stroma. The bone-marrow was replaced by newly formed bone lamellae surrounded with tumor tissue. The arrangement of the tumor cells in alveoli and tubules was similar to that of an adenomatous growth. This, together with the large-sized

Fig. 2.—Photomicrograph of a longitudinal section of a blood capillary coursing between single and double rows of endothelial tumor cells. Magnification: Distance, 12 inches; ocular, 4; objective, 3.

cylindrical and polygonal flat cells, abundantly present in the sections, easily gave an impression of a carcinoma. However, a closer and more careful study of areas where the tumor tissue was less mature and with fewer degenerative changes, revealed an entirely different picture. There were areas where the origin of the tumor cells could be traced to normal endothelium of the blood vessels. There were blood vessels showing on one side of their wall normal endothelium, while on the other side the endothelium had begun to proliferate and to pile up toward the lumen. The endothelial cells had grown larger. The nuclei, the majority of which were single, had become enlarged and vasicular; the nucleoli were very prominent. The cytoplasm had swollen but still retained the weak affinity towards basic and acid stains. Because of the

enlargement of the cells and their mutual pressure, the cells had become more or less polygonal and often cylindrical. We frequently found tumor cells containing two or more nuclei. On closer examination, it was not difficult to see that the majority of these giant cells were groups of tumor cells with scarcely visible cell boundaries. The true tumor giant cells which were present were easily distinguished from the remaining osteoclasts.

In many instances, the tumor cells lay against the capillaries, while the latter coursed between single or double rows of these cells. It was possible to trace the development of these tubules of tumor cells from blood vessel endothelium, the newly formed cells protruding from the vessels and forming rows in the

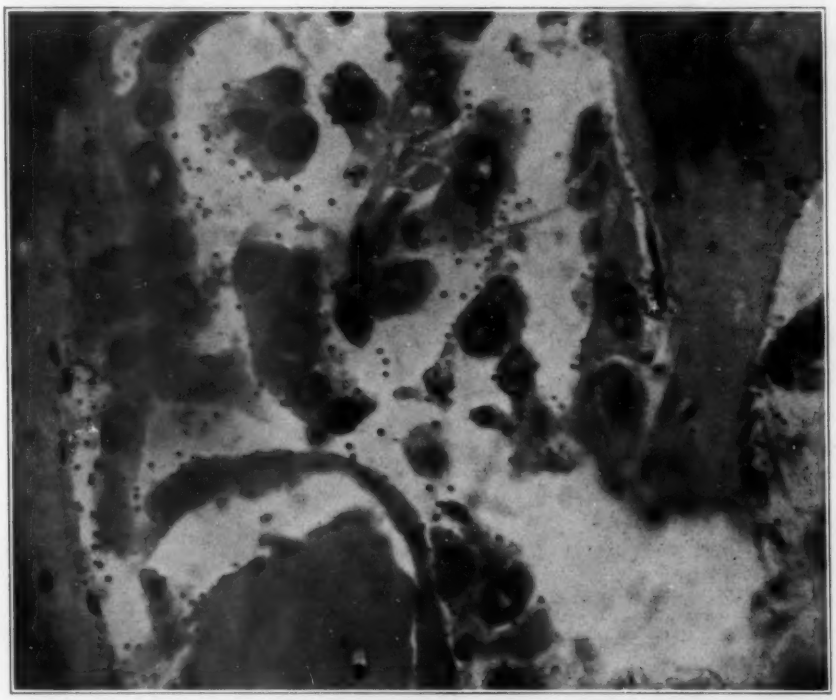

Fig. 3.—Photomicrograph, showing various transitional forms of the endothelial tumor cells. Magnification: Distance, 12 inches; ocular, 4; objective, 6.

scanty surrounding tissue. These long rows of tumor cells seemed to line spaces which first contained blood. Later on, these tubules had become obliterated so that no blood elements could be found in them. Longitudinal and cross sections of such obliterated tubules markedly resembled an adenomatous growth. These strands of tumor cells were more or less intimately connected with the surrounding stroma and not separated from it by means of endothelial cells lining the connective tissue spaces, a condition always seen in the case of carcinoma.

The mediastinal and mesenterial lymphnodes show an extensive involvement. The morphology of the tumor cells in these glands, as well as in the miliary

metastatic nodules in the lungs, is the same as in the bones. The transitional forms of the endothelial tumor cells are very distinct in metastatic nodules in the lymph nodes.

These results of the microscopic examination led me to the final diagnosis of primary multiple endothelioma of the skeleton.

Fig. 4.—Fibular tumor with distinct new bone formation.

## COMMENT

There is considerable discrepancy in the opinions of authorities on the recognition of endotheliomas, in general, and endotheliomas of bone, in particular, and the literature is so abundant that, as an illustration,

I shall merely quote two authors. Ribbert [2] says: "In no instance has it been proven that a so-called endothelioma originated from the endothelium." "The knowledge of endothelioma of bone reached a stage which calls for definite recognition of the disease:" says Ewing.[3] In view of this discrepancy of opinions and of the possible opposition to this diagnosis, I sent sections of the tumor to ten leading pathologists in the United States, Germany and Russia. The answers I received contained the following classifications: Five diagnosed the tumor as endothelioma

Fig. 5.—Circumscribed tumor on the left tuber ischii. The vertebrae are not involved.

of bone; one made the categorical diagnosis of metastatic carcinoma; one was inclined toward carcinoma, and three toward endothelioma. None of the men consulted had seen the roentgenograms before making his diagnosis.

The cause of the marked discrepancy in the literature is mainly due to the characteristic morphologic peculiarities of endothelioma of bone.

2. Ribbert, Hugo: Geschwulstlehre, Ed. 2, Bonn, 1914, p. 245.

3. Ewing, James: A Review and Classification of Bone Sarcomas, Arch. Surg. **4**:487 (May) 1922.

The peculiar arrangement of the tumor cells surrounding the blood vessels in groups, simulates, in sections, alveoli and tubules of a carcinomatous growth. However, with a more careful study of the tissues,

Fig. 6.—Tumor on the left scapula. Note the prominent new bone formation and the sharp contours of the tumor.

there usually can be demonstrated fine points which alone can decide the diagnosis. These, in the present case, are:

1. The tracing of the tumor cells to the endothelium of the blood vessels is possible. The proliferation of the endothelium by itself is also encountered in carcinoma, when tumor cells fill the lymphatics and blood channels. It is then a normal reaction to the presence of a foreign

tissue. But in carcinoma, the difference between the proliferating endothelium and the carcinoma cells is so obvious that it cannot be overlooked.

2. The intimate attachment of rows of tumor cells (tubules) to the surrounding stroma is noticeable. In a carcinomatous growth, the groups of tumor cells are separated from the surrounding stroma by means of endothelial cells lining the invaded connective tissue spaces. Ribbert,[4] who is most radically opposed to the recognition of endo-

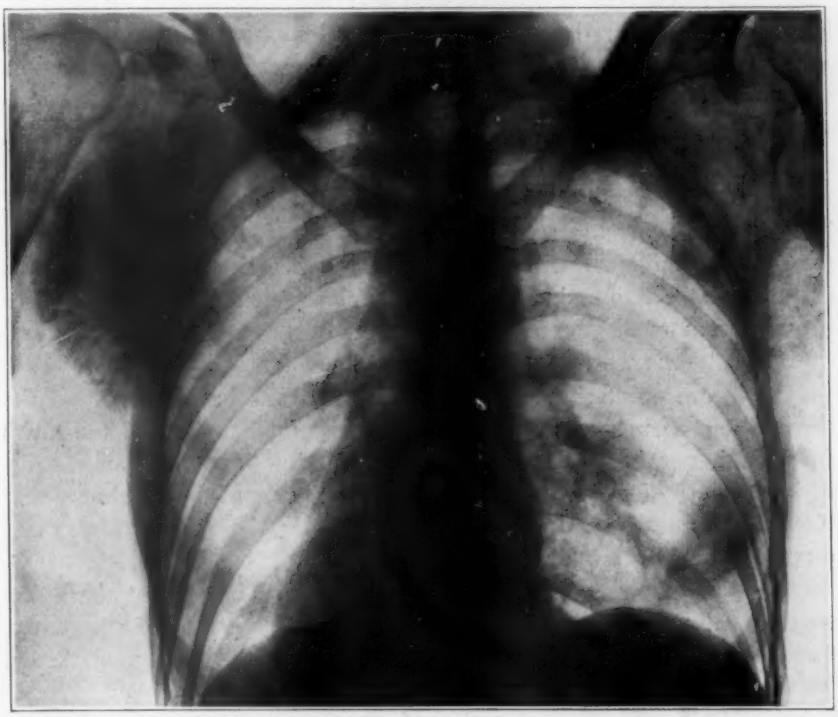

Fig. 7.—Roentgenogram of the chest showing an extensive involvement of the ribs.

theliomas, insists that "das Festhalten an der bindegewebigen Unterlage" is not a characteristic feature of endothelial cells because there are instances when endothelial cells readily loosen from the underlying stroma in the walls of lymphatics and lymph sinuses of lymph nodes. It is obvious that such exceptional cases do not negate the fact that, while in carcinoma the groups of tumor cells can be readily brushed out from their supporting stroma, in tumors called endothelioma, there is an

4. Ribbert, Hugo: Geschwulstlehre, Ed. 2, Bonn, 1914, p. 238.

intimate organic connection between the tumor cells and the surrounding stroma.

3. Apart from the apparent giant cells, true giant cells are to be found containing two or more nuclei. Giant cells are often found also in metastatic carcinoma of bone. The question of their rôle there is a disputable one, but all agree that they are the large, polynuclear osteoclasts of normal bone-marrow and that they do not belong to the tumor cells. In the present case, the giant cells are typical tumor cells and the only difference between them is their size and number of nuclei. That these giant cells are not osteoclasts was ascertained also by such an authority on normal bone-marrow as Dr. Maximow. As a matter of fact, the finding of giant cells is frequently reported in cases of tumors diagnosed as endothelioma of bone.

4. The location of the tumors does not indicate metastatic carcinoma. Handley asserts that the favorite site of metastatic carcinoma is located above the elbows and knee-joints. Recklinghausen pointed out that the bones under constant stress are most liable to become involved in metastatic carcinoma. In other words, the favorite site of carcinoma is the axial skeleton and not the appendicular skeleton. Of twenty-three cases of tumors diagnosed as endotheliomas and reported in the literature, twenty-one were located in the appendicular skeleton.

One more point should be mentioned. It has been stated that the involvement of mediastinal and mesenterial lymph nodes and the lungs favors a diagnosis of metastatic carcinoma and not endothelioma. Crile, in his review of reported cases of endothelioma of bone, shows that visceral metastases occurred in sixty-three cases, 6 per cent. of all cases reported. Ewing is of a similar opinion.

Turning now to the roentgenograms, we notice, in the first place, that we are dealing with a malignant tumor. The multiplicity of the tumor, its nonperiosteal origin and the age of the patient point to malignancy. Primary sarcoma can be excluded on the basis of its multiple character alone. The nonmedullary location of the tumor, the absence of diffuse absorption of bone, and the presence of new bone formation are against a diagnosis of multiple myelomas. So the diagnosis must rest between metastatic carcinoma and multiple endothelioma of bone. The roentgenologic features of the metastatic carcinomas are different according to whether they are osteoclastic or osteoplastic. In the osteoclastic type, the bone is destroyed without any expansion of the shell of the bone; the cortex is swept away and consequently does not throw any shadow whatever. In the osteoplastic type, the tumors are not limited locally and there is found a mottled appearance of the whole bone. In the present case, the tumors are distinctly limited, and the shaft shows nothing but a readily explainable atrophy of bone. As I mentioned

above, carcinoma seldom invades the appendicular skeleton without previous or additional involvement of the axial skeleton. Here, we see the tuber ischii involved during a period of eight months, while the dorsal and lumbar vertebrae do not show any roentgenologic evidence of involvement. Finally, the presence of large osseous tumors in the involved bones also speaks against carcinoma. In osteoplastic carcinoma, it is only occasionally that the external surfaces show evidence of disease, and then the manifestation is generally a superficial, irregular deposit of new bone. Large, clinically detectable tumors are unknown in osteoplastic carcinoma. Such tumors are encountered in exceptional cases where the diagnosis is osteoclastic carcinoma, but then they do not throw any shadow since they do not contain any bone.

Thus, the roentgenologic data confirm the diagnosis made above. As I mentioned, the roentgenologic study of endotheliomas of bone has been almost wholly neglected. In fact, Ewing is the only author who paid attention to this side of the diagnosis of endotheliomas. Ewing discusses the roentgenologic features of a primary endothelioma of bone. He states that in multiple endothelioma and in solitary diffuse endothelioma there is a diffuse absorption of bone "without a trace of bone production." [5] However, Figure 3 of his paper reproduces a roentgenogram of an ulna with diffuse endothelioma showing a very marked bone production. A rare case of endothelioma shown me by Dr. Phemister in his laboratory had prominent signs of bone production. Thus, in the three cases of primary endothelioma of bone which have been studied roentgenologically, Ewing's, Phemister's and the present case, there is a definite bone production. I certainly do not mean to claim that in endotheliomas there is always bone production, but I think that the reverse is not true. The tumor tissue invading the involved area of the bone leads to a swelling and diffuse enlargement of the latter. Such a bone feels very soft; it throws a shadow less dense but of a larger size because the amount of bone tissue is not less and indeed may be even more than before involvement. These considerations seem to emphasize that roentgenologic study is indispensable in the determination of bone destruction or production in more or less extensive bone tumors. Not only the density of the shadow, but also the size of the shadow is important for the conclusion. I am far from the opinion of those who claim, like Nichols, that "We shall be impressed by the ease with which bone tumors may be diagnosed roentgenologically by means of this simple classification," [6] but still I think that we can bring in more light in this dark field of endo-

5. Ewing, James: A Review and Classification of Bone Sarcomas, Arch. Surg. **4**:491 (May) 1922.

6. Nichols, Bernard H.: Roentgen Diagnosis of the More Important Tumors of the Long Bones, Surg., Gynec. & Obst. **35**:301 (Sept.) 1922.

thelioma of bone by paying more attention to the roentgenologic study of the tumors. Unfortunately, the results of histopathologic study of bone tumors have so far proven to be unsatisfactory, especially in complex cases where the diagnosis is of a special interest to the surgeon. One can hardly ever diagnose, with certainty, a complex case by roentgen ray alone; rarely, histologically. Only a combination of both, with the experience of the surgeon, will solve this problem.

# ABSENCE OF SACRUM

## WITH REPORT OF A CASE, AND A REVIEW OF THE LITERATURE*

S. F. STEWART, M.D.

LOS ANGELES

Absence of the sacrum is an extremely rare condition. Only six cases have been found recorded or referred to after a thorough search of the literature. The appearance of this abnormality in a clinic is worthy of note.

### REPORT OF CASE

*History.*—A Mexican boy, aged 6 years, admitted to the Children's Hospital in October, 1922, had no control of the bladder and bowels. He had double club feet and some deformity in the region of the lower spine at the time of birth. Tenotomies were performed on the feet when the child was 2 days old, and a moderate correction was obtained.

The birth of the patient had been normal at term. He sat up alone at 18 months, and walked at 2 years. Dentition was normal. He had had no contagious diseases or other illnesses. He talked plainly, was very active on his feet and was said to be of normal mentality.

The father and mother were well, as were two brothers and three sisters. There had been two stillbirths, and one child was killed in an accident.

*Physical Examination.*—The child was of about normal size, but was not particularly bright looking. He was undernourished and underdeveloped. Aside from a slight blepharitis, there was nothing worthy of note in the general examination. The child stood without aid. The left foot rested normally on the floor, while the right was held in a slight equinovarus position. He also walked without aid, and with no peculiarity other than that due to the equinovarus.

The spinous processes could be distinctly felt down to and including the fifth lumbar vertebra. There was no scoliosis. At about the level of the third lumbar vertebra and extending down as far as the fifth, there were two swellings, one on either side of the spinous processes. These swellings were symmetrical, about 4 cm. long, 1.5 cm. wide and 0.5 cm. in height. They were most marked when the child was standing and were definitely fluctuant. The skin over them was normal in character. No sacrum could be palpated, but a narrow space could be felt between the ilia. The pelvic iliac crests measured 11.0 cm.; trochanters, 18.0; symphysis to tip of fifth lumbar vertebra, 11.5; left posterosuperior spine to right anterosuperior spine, 13.0; right posterosuperior spine to left anterosuperior spine, 13.0; posterior superior spines, 2.0; anterior superior spines, 14.0; tuberosities of ischia, 2.5.

There was little resistance by the sphincter to the examining fingers. The rectum was large and filled with feces. Posteriorly, only the skin and rectal wall could be felt for quite a distance; then the ilia came close together, and across the intervening space dense ligaments could be felt; but no bone was

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* From the orthopedic clinics of the Children's Hospital, Los Angeles, and the San Bernardino County Welfare Bureau.

felt in this region. At the extreme depth of digital examination, what seemed to be the inferior surface of the body of the fifth lumbar vertebra could be felt, but could not be traced with distinctness.

There was a real shortening and lack of development of the right lower extremity as compared with the left. Both lower legs were much less developed proportionately than the thighs. The feet were of equal length. The left foot was practically normal in shape and could be flexed actively both dorsally and

Fig. 1.—Anterior-posterior roentgenogram, showing the absence of the sacrum and coccyx. The enormous development of the spinous process of the fifth lumbar vertebra, and the apparent articulation of the transverse and spinous processes with the ilia may be noted.

toward the sole to a moderate degree. The toes could be moved slightly, and the great toe was held in slight dorsiflexion. The right foot was held in slight equinovarus, and the Achilles' tendon was contracted. Only the slightest amount of active and passive motion in the foot was obtainable.

*Neurologic Findings.*—As nearly as could be determined, sensation in the lower leg was somewhat confused. No clonus, Babinski or plantar reflexes were

obtained. There was total incontinence of both bladder and bowels. The slightest irritation caused by walking or standing was likely to precipitate a mass reaction of the bowels and bladder.

*Laboratory Findings.*—The urine was not remarkable. The Wassermann test of the blood was negative.

Roentgen-ray examination revealed complete absence of the sacrum and coccyx. The ilia were articulated with the transverse processes of the fifth lumbar vertebra, and apparently also with the spinous process. The pelvic inlet

Fig. 2.—Slightly oblique lateral view, showing the absence of the sacrum and coccyx, and a complete modification of the sciatic notches.

was much decreased in transverse diameter and appeared to be oval in shape.

*Other Findings.*—The character of the swellings on either side of the spinal column aroused the suspicion that we were dealing with a bilateral spina bifida of a semioccult type. At a subsequent examination, a fairly large needle was inserted over each of these swellings. After passing through the skin and subcutaneous tissue, the needle was plunged through a dense membrane into what appeared to be a sac, and on the anterior surface came in contact with bone. However, no fluid could be aspirated from either side. The manner in which

the needle entered the dense membrane reminded one of the way in which a needle enters the spinal canal. It was therefore an uncertainty as to the nature of the fluctuant swelling on either side of the spinous processes.

A diagnosis of absent sacrum and coccyx, total incontinence and bilateral equinovarus was made.

Clifford White [1] presented to the obstetric and gynecologic section of the Royal Society of Medicine the case of a fetus, stillborn at term, which had a congenital absence of the sacrum. A tracing of the roentgenogram is shown in Figure 3. The vertebrae were very irregular down to the twelfth dorsal, below which were only two cartilaginous nodules, the left articulated with the left ilium, the right connected with the vertebral column only by ligaments. The ilia were in contact posteriorly. Associated with these defects were umbilical hernia, absent anus and rectum, horseshoe kidney, obliterated left ureter, right undescended testicle, absent vas and double talipes equinovarus. From the lower dorsal region, a nerve ran down to the front of the thigh, which represented the anterior crural and great sciatic nerve. The muscles of the thigh were normal in appearance.

Wertheim [2] reported the case of a full-term living girl whom he was called to see just after birth. She was one of dichorionic twins; the other was found macerated. The infant presented a round, elastic mass the size of the child's head, occupying the region from the last lumbar vertebra to the anus. This tumor, which was covered with skin, was scarlet and was fluctuant in places. The skin of the perineal body was raised like a blister. Some old, thick blood was aspirated from the tumor without affecting its size. Death occurred on the eighth day. At necropsy, the tumor-like mass was found, covered by a thick layer of subcutaneous fat, beneath which was a thick atheromatous mass, ventral to which lay a cyst filled with thick bloody serum. The walls of this cyst were in direct contact with the rectum. The ilia were in close contact with the atheromatus mass, and were not completely ossified. The perineal sac forming the pouch of Douglas was not recognizable. The spinal column ended with the fifth lumbar vertebra. No other deformities are mentioned. Wertheim believed that trauma, which occurred at about the fourth month, was the causative factor.

Deschamp [3] has reported the case of a baby girl, several weeks old, who had a tumor in the sacral region, the size of a large apple. The tumor was firm. Its surface was irregular and covered with a thin, bluish skin which showed some ulcerations. It was considerably augmented in volume under abdominal compression. In addition, the same

1. White: Proc. Roy. Soc. Med. **4**:278, Part 2, 1911.
2. Wertheim: Monatschr. f. Geburts. u. Fraunkrankh. **9**:127, 1857.
3. Deschamp: Ann. orth. et chir. prat. **7**:367, 1894.

strains caused a forward projection in the region of the buttocks of what was demonstrated to be intestinal loops. The general condition of the child was excellent, and there was no apparent paralysis. Three hundred cubic centimeters of a yellowish fluid was withdrawn, permitting a collapse of the tumor. No resistant surface was found beneath the tumor.

A diagnosis of spina bifida with complete absence of the sacrum was made. The tumor later refilled, and an attempt was made to aspirate it. On opening the sac, it was found to be a hernia of the spinal meninges, which issued from the spinal canal of the last lumbar vertebra. Anteriorly, the peritonealized intestinal mass herniated into the wound. The meninges were incised and found to be very much thickened; and an

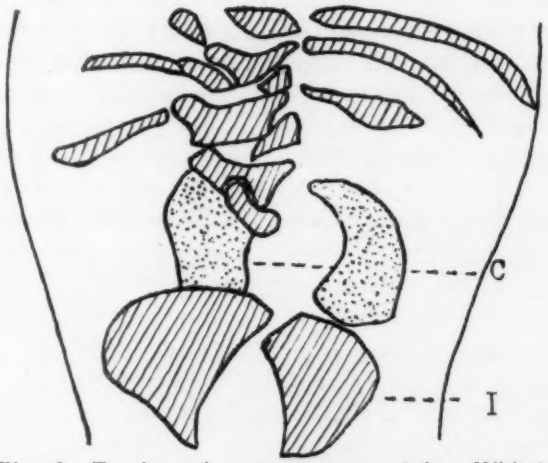


Fig. 3.—Tracing of roentgenogram (after White).

attempt to close the spinal canal with meningeal flaps was made. Three months later, the child was in good condition, but still exhibited the hernia in the region of the buttocks.

White [1] mentions the fact that Ballantyne, Luton, and Litzmann have described similar cases; but a most careful search of the Index Medicus, and Surgeon General's Catalogue has failed to reveal the location of these reports. He also states that the condition usually occurs in one of twins and is associated with equinovarus.

These deformities can result only from a lack of development during the first month of embryonic life. In the case that is here presented, it would seem that approximately the last ten primitive segments failed to develop. This condition cannot be a manifestation of atavism; neither can it be considered as one of the forerunners of the next stage of human evolution. If it were atavistic, we would expect the presence

of a primitive tail. Sir Arthur Keith,[4] in his Hunterian lectures, points out that in the pronograde monkeys there are seven lumbar vertebrae and a very small sacrum; and as one follows the ascent from the pronograde, through the orthograde to the plantigrade, there is a continual shortening of the lumbar spine owing to the increased sacralization of the erstwhile lumbar vertebrae, thereby augmenting the area of the sacrum in the plantigrade. It is his belief that this is brought about to give a greater base for the attachment of the more frequently used erector spinae muscles.

To the obstetrician, the case here presented represents the most marked form of the Robert or transversely contracted pelvis, but it is probable from the deformities that accompany it that its practical significance to them would be small. To the orthopedist, the almost universal association with equinovarus is worthy of note.

As in many other developmental defects, nothing is known of the cause, and nothing has been suggested as to treatment. The necropsy findings of White would lead one to the belief that marked rearrangements of the peripheral nerves might seriously interfere with the treatment of the associated deformities.

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4. Keith, A.: Brit. Med. J. **1**:451, 499, 545, 587, 624, 669 (March 17-April 21) 1923.

# A REVIEW OF UROLOGIC SURGERY

E. STARR JUDD, M.D.; ALBERT J. SCHOLL, M.D.
LINWOOD D. KEYSER, M.D.
AND
GORDON S. FOULDS, M.D.
ROCHESTER, MINN.

## KIDNEY

*Surgical Technic.*—Anterior pyelotomy was first described by Kapsammer, who claimed that it was not followed by fistula. Rosenstein [1] claims that there is no more risk of injuring vessels than by the posterior route. Aberrant vessels, or branches, commonly pass behind the renal pelvis. In some cases the entire upper branch of the renal vein may pass posteriorly to the pelvis. Rosenstein believes that the anterior approach to the kidney is the natural one; the pelvis is reached by retracting or separating the artery and veins. The injury to the kidney is less with this approach than with a posterior incision. A better view is obtained of the pelvis, especially in the obese patient with a short, thick torso, and if there is perinephritis or peripyelitis. There is less hindrance to the outflow of urine following an anterior incision, and the danger of forming a fistula is less.

Zondel [2] employs three methods of entering the renal pelvis:

1. The usual, or posterior pyelotomy incision which gives the best exposure and is easier to close; it is made parallel to the ureter, and extends almost up to the renal parenchyma. Zondek sometimes uses a transverse incision in the pelvis and occasionally the two are combined.

2. The anterior incision developed by von Illys is employed if the vascular pedicle is short, or if the anterior surface is more accessible. In corrosive specimens of the kidney it was found that fewer small blood vessels were cut than with the anterior incision. After closure of the anterior incision, the large vessels fall together over the incision, tending to close the opening. The anterior incision is necessary in certain cases of anomalies, especially horseshoe kidneys, and other types in which the complete pelvis faces anteriorly.

3. An inferior pyelotomy is performed for anatomic reasons, if the remaining portion of the pelvis is covered, or if the inferior border presents easily. Zondek makes an inferior pyelotomy to remove stones in the inferior major calyx, and for stones lodged in the lower part of the pelvis.

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1. Rosenstein, P.: Die Pyelotomie anterior, Ztschr. f. urol. Chir. **12**:269-289, 1923.

2. Zondek, M.: Pyelotomie, Ztschr. f. urol. Chir. **12**:163 (March) 1923.

[Ed. Note.—If the patient is in good position for delivering the kidney, and the incision is sufficiently large to permit good exposure, it is only rarely that the posterior incision is unsatisfactory. With the anterior incision, injury to the large vessels is quite probable, either with instruments, or during the removal of irregular stones. Pelviolithotomy has apparently not been the operation of choice in Europe for many years. In the early days when gauze drainage was commonly employed, postoperative fistulas were common. Consequently, nephrolithotomy was often the operation of choice, and is still used to a much greater extent than in this country.

With a new method of approach, anterior pyelotomy, gauze drainage is not employed, consequently fistulas are rare. Posterior pelviolithotomy, very properly, has been established as the operation of choice in this country for the removal of renal stones, and will probably be employed until some decidedly more satisfactory procedure is offered.]

Gibbon [3] reviews certain phases of renal and ureteral surgery as they have presented themselves in his practice during the last fifteen years. He believes the abdominal route to be preferable to the lateral in cases of large tumor. In case drainage is necessary, the renal fossa may be drained posteriorly through a stab wound, and the peritoneum over it closed. In the removal of renal stones, Gibbon advocates pyelotomy with extension of the pelvic incision into the renal tissue if necessary (Eisendrath's enlarged pyelotomy). He believes that leakage is insignificant if the incision has been clean-cut and gauze drainage is not employed. In dealing with bilateral stones he emphasizes renal function. If renal elimination is poor on both sides, any operation will be risky. He usually operates on the side of poorer function first. With ureteral stone on one side and renal stone on the other, effort should first be made to make the ureteral stone pass. Nephrectomy for stone is rarely indicated and should never be done unless the kidney is hopelessly damaged by infection or injured by manipulation, chiefly because of the possibility of stone in the other kidney. Again, the potential function of the kidney, after removal of the stone and drainage, is difficult to determine.

In the removal of stones from the lower ureter, Gibbon advocates cystoscopic methods when experienced technicians are available. Contraindications are stones more than 2 cm. in diameter, acute infection, lodgment of the stone for from three to six months, and intolerance to cystoscopy.

The most satisfactory operative procedure for ureterolithotomy is the muscle splitting incision parallel to Poupart's ligament or a rectus

3. Gibbon, J. H.: Technique of Renal and Ureteral Surgery, Ann. Surg. **38**: 641-648 (Nov.) 1923.

sheath incision. The peritoneum is reflected toward the pelvis until the iliac vessels and the pelvic wall below them are exposed. The ureter adheres to the peritoneum and is reflected with it. It is most easily identified by palpating the stone or by searching for the point where it crosses the iliac vessels. The stone should be localized before opening or mobilizing the ureter. If this cannot be done, a button-hole is made in the peritoneum, a finger applied, and the stone loosed and pushed up with the ureter until it can be readily felt in the extraperitoneal wound. Then it may be excised, the ureter closed, and the peritoneum sutured.

In females with stones lying low in the ureter, an assistant's finger in the vagina may help in bringing the stone into view in the extraperitoneal wound. A dilated ureter is easily identified and in these cases the ureter may be opened above the stone, which may then be removed by the Mayo common duct forceps. Gibbon does not place sutures in the ureteral wound, leaving it to heal without surgical closure. Little or no leakage follows.

Beer [4] describes a method of closing nephrotomy wounds by passing interrupted double chromic sutures through the two sides of the incision, the distal ends being tied. Before pulling the distal loop close to the renal surface, a piece of fat is placed under the loop, and the suture drawn tight. On the other side of the organ, where the needle has been brought out between the two loops of chromic catgut, a second piece of fat is placed, and over this the two ends of the catgut are tied. The knot can be pulled up as tight as is necessary to control the bleeding, and as the stitch is underpinned on both sides, there is little chance of cutting into the parenchyma.

[ED. NOTE.—While pyelotomy has largely displaced nephrotomy in certain selected cases it is necessary to incise the kidney to remove large calculi or smaller stones embedded in minor calices. The suture of the kidney, under such conditions, has remained a surgical problem of great importance. To minimize the area of anemia and secondary infarction necrosis, numerous types of sutures have been used. Brodel's triangular suture, Stellwagen's through-and-through sutures, underpinned with catgut, and Hagenbach's through-and-through sutures, underpinning with fat in much the same manner as that described by Beer, have been advocated. Beer's technic is simple, and theoretically seems to offer an opportunity for maximal hemostasis with minimal destruction of parenchymal tissue.]

Bazy,[5] in 1914, first advised an anterior transverse abdominal extraperitoneal incision for the exposure of the kidney. In a recent review,

4. Beer, E.: The Use of Fat to Prevent Sutures Cutting Into Parenchymatous Organs by Underpinning, Surg., Gynec. & Obst. **37**:694-695 (Nov.) 1923.

5. Bazy, L.: Chirurgie die rein, Soc. de Chir. **48**:1318-1328, 1923.

he reports forty-three cases operated on through this incision. The incision, which may be either a little above or a little below the level of the umbilicus, is made through the anterior abdominal muscle, somewhat similar to that occasionally employed at one time for exposure of the gallbladder. The peritoneum is not opened, but retracted toward the middle line. This gives access to the renal pedicle and permits a view of the great vessels. Metastases may be readily discovered, and the vascular pedicle may be ligated under the eye. The upper ureter may also be examined. Bazy favors this method of exposure rather than the lumbar, which, he states, opens on the convex border and inferior portion of the kidney, too far away from the pedicle, so that the pedicle is clamped last and blindly.

[ED. NOTE.—With an accurately placed posterolateral incision, it is only rarely that access to the renal pedicle is not readily obtained. With the patient satisfactorily placed, the kidney delivers very freely into the lumbar incision, readily permitting examination and operative procedures. The position of the lumbar incision is ideal for dependent drainage, and subsequent incisional hernia is very rare.

Injury to the peritoneum more frequently occurs in the abdominal exposure, and it is more necessary to preserve the integrity of the strong anterior abdominal muscles than the lateral body muscles, which readily unite.]

Jianu [6] discusses the bleeding following renal operations, which may either be primary from one to two days after operation, or secondary from six to twelve days after. Rafin believes that the bleeding is the result of infection; a number of other observers consider that it is caused by renal infarction. Legueu has observed sixteen cases of severe postoperative bleeding which necessitated removing the kidney. In one case, bleeding was from a severed vessel. In four cases, it was from an infarct which developed in the incision through the renal cortex. In four cases, bleeding followed the tearing of an aberrant artery in doing a pyelotomy. Rehn believes that the bleeding results from circulatory and secretory disturbances which upset the physiologic balance of the kidney, resulting in abnormal drainage conditions. The author believes that the bleeding which is most commonly seen after nephrotomy results from trauma to the intrarenal vessels. He suggests a method of controlling hemorrhage in extensive nephrotomy incisions by placing a series of overlapping sutures in the renal cortex before the kidney is closed. These sutures enter the cortex of one side of the kidney, pass down toward the hilum and into one of the calices, or pelvis, then return about 1 cm. farther along on the same side of the kidney.

6. Jianu, A.: Die postoperative Nierenbluting, Ztschr. f. urol. Chir. **14**:38-45, 1923.

On ligation, each suture controls a definite unilateral segment of renal tissue. Similar sutures are placed in the opposite half. The kidney is closed and drained. The sutures used for closure of the nephrotomy incision are also used to fix the kidney to the body incision.

[ED. NOTE.—Multiple sutures of this type entail a long operation, leave a large amount of catgut in the kidney, and are unquestionably followed by partial destruction and necrosis of the renal parenchyma. Hemorrhage and infection are the most serious complications which follow extensive nephrotomy of this type. Infection is a much more common cause of secondary nephrectomy, and its extension is greatly facilitated by trauma, foreign bodies, and necrosis resulting from multiple ligatures.]

Gayet [7] performed heminephrectomy for tuberculosis in horseshoe kidneys in two cases; one case was complicated by multiple stones. The patient was large and fleshy, and the kidney was hydronephrotic. The vascular pedicle was clamped and ligated. The clamps were left in place after the operation, and removed on the fourth day. The isthmus, which was thick and parenchymatous, was sectioned and sutured over. On section, the kidney was found to be almost completely destroyed with caseating tuberculosis. Five months later, the patient was in good health, but the wound was still draining.

[ED. NOTE.—Anomalous, distorted kidneys apparently offer favorable soil for the growth of the bacilli of tuberculosis. Ten cases of heminephrectomy in horseshoe kidneys for tuberculosis have been reported; seven patients recovered. In Gayet's cases a fistula formed, probably because of extension of the infection into the isthmus. Carlier reported a case in which tubercles were found in the band of tissue connecting the two kidneys.]

Eisendrath and Portis [8] report experimental ligations of the renal vessels in dogs. The kidney in the ligated side underwent atrophy, cessation of function, and degenerative parenchymatous changes, such as hemorrhagic infarction, extensive necrosis of cellular elements, and diffuse infiltration with blood cells. Clinically, they have applied preliminary ligation of the renal vessels in a case of hypernephroma, in which, because of extensive hemorrhage at the time of attempted nephrectomy, operation had to be abandoned. After preliminary ligation of the vessels by the transperitoneal route, a secondary nephrectomy was performed later with a comparatively bloodless technic. A second patient had a long standing suppurative condition around a pyonephrotic kidney. The right renal vessels were ligated transperitoneally, and

7. Gayet: Rein en fer a cheval tuberculeux, Lyon med. **132**:150-151, 1923.

8. Eisendrath, D. N., and Portis, B.: Primary Ligation of the Renal Vessels as an Aid to Nephrectomy, J. Urology **11**:143-157 (Feb.) 1924.

several weeks later the pyonephrotic kidney was removed without bleeding from the kidney or pedicle. The technic of abdominal approach to renal vessels is reflection of the parietal perinoteum along the outer border of the duodenum on the right, and along the area just beyond the duodenojejunal juncture on the left.

[ED. NOTE.—The preliminary ligation necessitates an intra-abdominal operation on a patient who already is a poor operative risk. Operation in several stages, especially in patients of poor vitality, seems to be coming into popularity, as is evidenced by the good results that are being reported in thyroid, chest, and colon surgery. Clinical experience has proved that this technic is of definite value in a restricted group of cases in which primary nephrectomy would be hazardous.]

Molesworth [9] reports a case of traumatic rupture of a hydronephrotic kidney. Nephrectomy was performed successfully. He was able to trace eleven additional cases in the literature. In none of these was a correct preoperative diagnosis made. The rupture is usually radial, and the peritoneum frequently torn. This is a strong argument in favor of opening the abdomen. Macalpine, in the same journal, reported a similar case in a child of eight years. The patient made a good recovery.

*Tumors of the Kidney.*—Riese,[10] in a review of the literature on renal malignancy, finds that in general the operative results are unfavorable. Paschen reported fifty-four cases of nephrectomy for carcinoma from the Hamburg Krankenhaus; 35 per cent. of the patients were alive three years after operation. Berlstein reported that only twenty-two of the patients operated on at Hocheneggs Clinic were alive and free from recurrence three years after operation. Riese reports ten cases; nephrectomy was performed in nine. There were two operative deaths, and only three patients are alive and well. One died three months after operation, and one fourteen months after. A third patient, with a large growth which was only partly removed and later was treated with roentgen rays, is alive and well six years after operation. Four patients had metastasis; metastatic growths were removed four times in two years from one patient, who died eight years after the first operation. Five of the operations were lumbar, and four transperitoneal.

[ED. NOTE.—Most series of cases include all cases in which operation has been performed during the last twenty or twenty-five years, and consequently in the period in which all renal operations were attended by a high operative mortality. Nephrectomy for cancer of the kidney is unquestionably followed by the highest early and late mortality of

9. Molesworth, H. W. L.: Traumatic Rupture of a Hydronephrosis, Lancet **2**:224-225 (Aug. 4) 1923.

10. Riese, H.: Beitrage zur Chirurgie des Urogenital systems, Ztschr. f. urolog. Chir. **12**:334-352 (March) 1923.

any operation on the kidney, but in reviews which deal only with operations performed in the last ten years, the immediate mortality is much lower, and does not reflect the poor prognosis offered by earlier cases.]

Schwarz [11] reported several interesting renal tumors before the Berlin Urologic Society. In the first case, the removal of a carcinoma of the left kidney was followed by normal convalescence during the first two weeks, but shortly afterward a fecal fistula developed. The wound and abdomen were explored, but nothing unusual was found; the renal area was packed with gauze. The patient improved somewhat, but died six months after operation with extensive glandular enlargement in the right inguinal and right axillary regions. In the second case, the left testis had been removed, apparently for tuberculosis. This was followed several months later by a pain in the left loin. It was thought that the condition was probably left renal tuberculosis, but pyelography revealed an attenuated, distorted renal pelvis that was strongly pushed away from the median line, a typical picture of renal tumor. On reexamination of the excised testis, it was concluded that it was malignant and that the kidney was secondarily involved. An enlarged gland in the neck was removed and found to be sarcomatous. The patient was treated by roentgen ray. In another case, pyelography revealed a broadened renal pelvis containing a filling defect. At operation, the kidney was found to contain a papillary tumor of the renal pelvis that had destroyed the upper half of the kidney. The kidney and 12 cm. of the ureter were removed through a single incision.

Foulds [12] reviewed 295 cases of tumors of the kidney seen at the Mayo Clinic. He classified 243 of these as carcinomas, according to Ewing. Pain, hematuria and tumor constitute a triad of cardinal symptoms which were present in 40.7 per cent. of this series; two of these symptoms occurred in 46 per cent. of the remainder. The type of hematuria was characteristically intermittent in approximately 60 per cent. of the cases. Recent varicocele is a common and important physical finding in the male. Cystoscopic and pyelographic evidence was found valuable in confirming the diagnosis in a very large proportion of cases.

A modification of Ewing's classification includes: (1) Papillary adenocarcinoma, usually called hypernephroma (papillary carcinoma with clear cells, papillary adenocarcinoma with granular cells, and malignant papillary cystadenoma), and (2) alveolar carcinoma. The first main group includes 82.5 per cent. of the cases in which complete

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11. Schwarz, O.: Demonstration zur Chirurgie der Nierentumoren, Ztschr. f. Urol. **18**:133-136, 1924.

12. Foulds, G. S.: Carcinoma of the Kidney in the Adult, Bull. St. Michael's Hosp., Toronto **1**: No. 4, 1924.

data were available. The papillary carcinomas with clear cells constitute 59 per cent. of the tumors studied. These have the characteristic yellowish and brownish-yellow color, are well encapsulated, and often contain hemorrhagic areas. Microscopically, they are composed of a stroma of fine capillaries and of large clear cells arranged in rows or columns closely associated with the capillaries, the whole being distinctly papillary in formation. Papillary adenocarcinoma with granular cells are white, and usually only partially encapsulated. Their cells are smaller and more granular than those found in the clear celled growths. Malignant papillary cystadenomas are whitish, small, encapsulated growths, made up of a distinctly papillary stroma lined with small, well staining cuboidal cells. The alveolar carcinomas form rapidly growing soft tumors, without definite capsule, often invading the pelvis of the kidney, or breaking through the renal capsule. Histologically, they are made up of ovoid or rounded cells with large, deeply staining cells closely packed together, there being very little evidence of cytologic differentiation.

Tumors located in the median portion of the kidney gave a slightly better prognosis than polar tumors. Invasion of the renal vein by the tumor indicated a very poor prognosis in forty-five cases of venous involvement; nine patients died in the hospital, twenty-two have died since, five are untraced, and only nine are known to be alive. Certain patients with tumorous growths invading the renal vein may survive for a long time, however; one patient lived fourteen years after operation.

Wheeler [13] reports three cases of rare renal tumors. The first was an adenomyosarcoma in an infant, aged 8 months. In such cases, hematuria as a symptom is frequently absent. Attention is first directed to the presence of the tumor or to abdominal distention. As the growth enlarges, a train of pressure symptoms ensues. Transperitoneal removal of such tumors is advocated. Prognosis is poor, recurrence taking place in 80 per cent. of such cases within the first year after operation.

A case of squamous-cell carcinoma of the kidney pelvis is reported. The patient was a woman aged 56 years. The association of such tumors with renal calculus is emphasized. It is suggested that lithiasis and infection may incite a metaplasia in the renal epithelium to neoplastic growth.

A case of angioma of the left kidney in a man aged 57 is described. Hematuria was the presenting symptom and was profuse. The patient recovered, following nephrectomy. Angiomas of the kidney are very

13. Wheeler, Sir W. deC.: Some Renal Tumors, Surg., Gynec. & Obst. **38**: 143-149 (Feb.) 1924.

rare and are diagnosed only after nephrectomy, as a rule. There is great difficulty in differentiation from essential hematuria.

Broster [14] reports the case of a woman, aged 72 years, who had had a right nephrectomy for hypernephroma in 1914. She remained well for eight years, when she sustained a spontaneous fracture of the right femur; later, a swelling appeared at the site of the injury. The hip was amputated in February, 1923. The swelling of the femur proved to be a secondary hypernephroma.

[ED. NOTE.—This case is of interest because of the long interval between operation and recurrence. Albrecht [15] and Claremont [16] report cases in which the patients lived seven and ten years, respectively, after nephrectomy and then died from metastasis.]

Eggers and Felsen [17] report a case of papillary epithelioma of the renal pelvis in a male of 58 years. A history of long standing hematuria without much pain, negative roentgen-ray findings, and the excretion of pure blood from the left side with marked reduction of kidney function led to a diagnosis of renal tumor. Left nephrectomy was performed, and a papillary tumor of the pelvis with but little encroachment on the ureter was found. A secondary ureterectomy was done later. The ureter at this time was found to be uninvolved; the patient has been well four years since the operation.

Scholl [18] reports eight papillary tumors of the renal pelvis. All were histologically malignant. Three patients died from five to nine months after the operation, one of whom had a transplant to the ureter and another, a large secondary growth in the bladder. A fourth patient died from uremia four years after nephrectomy. Four patients are still alive, two are free from recurrence, one, two and one-half years, and the other four months after the removal of the diseased kidney and ureter. The remaining two patients have had repeated transplants to the bladder, requiring persistent treatment; at the present time both patients are well, one two years, the other, three years after the first operation.

Tumors of the renal pelvis are usually of epithelial origin and the majority are papillary in type. In the early stages the tumors are small,

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14. Broster, L. R.: A Case of Secondary Hypernephroma in the Femur with Secondary Fracture, Brit. J. Surg. **11**:287-294 (Oct.) 1923.

15. Albrecht, P.: Beitrage zur Klinik und Pathologischen Anatomie der Malignan Hypernephrome, Arch. f. klin. Chir. **77**:1033-1073, 1905.

16. Claremont, quoted by Ewing: Neoplastic Diseases, Philadelphia, Saunders, 1919.

17. Eggers, C., and Felsen, J.: Papillary Epithelioma of the Renal Pelvis, J. A. M. A. **82**:1415-1417, 1924.

18. Scholl, A. J.: Papillary Tumors of the Renal Pelvis, Surg., Gynec. & Obst. **38**:186-199 (Feb.) 1924.

often multiple, flat or thickly pedunculated and confined to the renal pelvis. They spread rapidly and extensively, involving the calices and sometimes the ureteral outlet. Obstruction of the ureteral outlet may cause an extensive pressure atrophy of the renal cortex. In the late stages the kidney becomes a distended, sacculated, often infected mass with complete loss of function. The renal pelvis does not offer a free space for growth like that of the urinary bladder, the pelvis is rapidly filled and the papillomatous masses become matted together under tension so that they bulge from the pelvis when the kidney is opened. The ureter is frequently involved in transplants, which are usually found in the normal ureteral constrictions. The individual papillomatous fronds are shorter and broader than similar growths in the bladder; there is a more extensive fusion of adjacent fronds and atypical cell masses are more often seen.

In the bladder, small papillomatous transplants may be found protruding from or surrounding the ureteral orifice. At times, multiple small transplants are scattered extensively over the mucosa of the bladder. Histologically, the majority of tumors of the renal pelvis, as well as the transplants in the ureter and bladder, are malignant. Clinically, the numerous extensions and the local recurrences make these tumors all malignant. Because of the frequency with which the ureter is involved and the repeated recurrences after nephrectomy, a complete nephro-ureterectomy is essential to insure even partial success.

Tumors of the renal pelvis are usually either papillary carcinoma or benign papillomas. Jacobsohn [19] reports a case of a man, aged 48, who had several attacks of severe hematuria and left renal colic. Roentgenography revealed a shadow of a renal stone in the region of the left kidney, which was not of the usual structure and density. At operation, a papillary tumor of the pelvis was found, which involved the upper pole of the kidney. The tumor had caused marked obstruction and dilatation of the renal cortex. The bleeding from the tumor had been so extensive that the patient was markedly anemic at the time of the operation.

[ED. NOTE.—The tumor reported by Jacobsohn was apparently benign. This is very unusual, as, in the majority of cases, examination of multiple sections almost invariably reveals areas of malignancy. Clinically, all tumors of the renal pelvis are malignant and readily extend to the ureter and bladder, requiring constant clinical observations in order to prevent extensive secondary deposits. In spite of a histologic examination, it is impossible definitely to determine after examination of the initial lesion what the outcome will be. A number

19. Jacobsohn, J.: Papillome des Nierenbeckens, Ztschr. f. Urol. **18**:137-138, 1924.

of reported cases of apparently benign primary lesions were quickly followed by malignant extensions or recurrence shortly after operation. Barth reported a benign papilloma of the pelvis with metastatic carcinoma in the regional glands. Reynes and Israel reported similar cases. Nephrectomy for benign lesions with development of generalized carcinomatosis is reported by Derwenko and Pantaloni.]

*Nephrolithiasis.*—Legueu and Fey [20] report a case of stone formation in an ectopic malformed kidney. The pelvis and calices faced anteriorly and there were a number of stones in the pelvis. The kidney contained six calices. The superior calix prolonged the upper border of the pelvis; there were two middle and three lower calices. This patient complained of attacks of colic localized in the anterior abdomen. During an attack, an ovoid mass was usually left in the abdomen, and the crepitation of stones could be made out. With right-sided abdominal tumor, crepitation and a feeling that the mass was directly under the abdominal wall, the condition simulated gallstones more than nephrolithiasis. Anteriorposterior and lateral roentgenograms were taken. The shadows were faceted, and the lateral view revealed the stones directly under the anterior wall; this together with marked mobility also suggested gallstones.

[Ed. Note.—Infection and stone formation are fairly common in abnormally shaped and misplaced kidneys and usually result from ureteral obstruction and deficient pelvis drainage. In ectopic kidneys there is usually almost complete destruction of the kidney when stones are present, which necessitates nephrectomy. Lateral roentgenograms have been made in this country by Nichols of Crile's clinic. In most instances, they readily differentiate renal from biliary disease, especially if pyelography is carried out. In Legueu's case, this was impossible, as the ureter was malformed.]

Rovsing [21] in twenty-nine years treated 716 cases of renal and ureteral stones. A bacteriologic examination of the urine was made in 589 cases.

Two hundred and seventy-six specimens were sterile, 197 contained *Bacillus coli,* fifty-four contained *Staphylococcus pyogenes* and fourteen contained streptococci; three, *Bacillus typhosis;* four, *Bacillus proteus* Hauser; one, gonococcus; two, bacillus of tuberculosis; thirty-three, *Bacillus coli* and staphylococcus; four, *Bacillus coli* and streptococcus, and one, *Bacillus coli* and *tuberculosis.*

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20. Legueu and Fey (Société Francaise d'Urologie): Calcul dans um rein malformé et en ectopie, Jour. d'urol. med. et chir. **17**:153-159, 1924.

21. Rovsing, T.: Ueber diagnose und Behandlung der Nierenstein auf Grund neun und zwanzigjahriger Erfahrungen, Ztschr. f. urol. Chir. **12**:358-384 (March) 1923.

Nephrolithotomy was performed in 256 cases, and thirty patients (11 per cent.) died; pelviolithotomy was performed in eight cases, and all the patients lived; nephrectomy was performed in seventy-six cases, and ten patients (13 per cent.) died; ureterolithotomy was performed in forty-five cases, and four (9 per cent.) died.

In eighty-two cases (14.2 per cent. of 716), the condition was bilateral. Twelve of the forty-four deaths occurred in this type of case. Of seventeen patients with anuria, six (35 per cent.) died. Twelve patients with uremia died; they had either chronic granular or toxic nephritis. Four patients died of pneumonia, four of pulmonary emboli, and two of hemorrhage.

In performing nephrolithotomy, Rovsing does not open the kidney widely, but cuts through the capsule, inserting a closed forceps down to the stone, which is held between the thumb and forefinger; the forceps is then opened, and the stone pulled through the cortex. Pelviolithotomy was performed in only eight cases. Rovsing believes that a pelvis incision gives poor drainage to an infected kidney, that the pelvis heals slowly, and that urinary fistulas are liable to occur. He performs a pelviolithotomy only in noninfected cases with single rounded stones. For ureteral stones, he makes an incision from the border of the erector spinal muscle toward the anterior superior spine, following the course of the iliac crest to about the middle of Poupart's ligament. If there is also a stone in the kidney, the ureteral stone is pushed up to the kidney and removed, either through a pelvic or a cortical renal incision. In simple ureterolithotomy, the ureter is closed with fine catgut sutures. Drainage is instituted in all cases.

Three hundred and thirty-one patients were treated by hygienic measures and the drinking of large amounts of distilled water.

Ward [22] reported a case of renal stone, weighing 555 gm. There was a smaller stone in the lower end of the ureter, and the kidney was completely destroyed. The stone was composed of ammonium magnesium phosphate and calcium phosphate. The patient had symptoms for twenty-three years.

[Ed. Note.—Stones of this size are now medical curiosities; however, a number have been reported recently. Shields reported a case in which the stone weighed 570 gm. Morrison reported two cases; in one, the stone weighed 960 gm. and in the other 1,200. In the second case, the opposite kidney also contained a small stone. Mylvagnum reported a case in which the extirpated renal mass weighed 2,160 gm., the stone weighing 1,440. This patient died fourteen hours after operation. Large stones, up to 200 or 300 gm., are not infrequently of the

22. Ward, R. O.: A Large Renal Calculus, Brit. M. J. **2**:563 (Sept. 29) 1923.

staghorn type, and may be bilateral or accompanied by a small stone on the opposite side. The extremely large stones, such as are considered here, are generally comparatively round or are slightly irregular on one side, the irregularity corresponding to the location of the obliterated and thinned out calices.]

Young [23] reports a case of stone in the upper third of the right kidney in a man aged 44. A pyelogram showed the stone to be situated in the upper calices, which were connected with the pelvis by such a narrow isthmus that removal of the stone by pyelotomy seemed unlikely. At operation, these findings were confirmed, the upper third of the cortex being thin and the remaining portion of the kidney apparently healthy. The upper third of the kidney was resected after removal of the stone through a nephrotomy incision. The diseased portion of the kidney, which was removed, contained the dilated calices and an infected area that might have led to further trouble with possible recurrence of the stone.

Crowell [24] reports a case of a woman, aged 27, who had a recurrence of cystin stone in both kidneys following two operations, one on the right and one on the left side, for the same condition. Alkalization of the urine, repeated lavage of the renal pelves with mercurochrome solution and normal saline at intervals of two to three days over a period of forty-five days caused a subsidence of infection and improvement in renal function. The patient was sent home, instructed to keep the urine alkaline by taking soda bicarbonate, and to return weekly for pelvic lavage. The stones shortly began to reduce in size, as shown by the roentgen rays, and disappeared entirely nine months later. The infection cleared up, and the urine remained free from cystin.

Since cystin is soluble in alkaline solutions, it is important to render the urine alkaline and to keep it so. Mercurochrome is alkaline as well as antiseptic and is also very penetrating. The author feels that these factors have been of prime importance in the handling of the case.

[ED. NOTE.—The solvent for stone has long been sought, and it is most gratifying that Crowell has demonstrated its possibility. First, we must know the chemistry of the stone, and second, we must introduce, directly or indirectly by feeding, the proper solvent for its chemical constituents. The mucosa of the urinary tract is irritated by alkalis and acids in sufficient concentration to dissolve the harder phosphate and oxalate calculi.

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23. Young, H. H.: Resection of the Kidney in Nephrolithiasis, Surg., Gynec. & Obst. **38**:107-111 (Jan.) 1924.

24. Crowell, A. J.: Cystin Nephrolithiasis; Report of Case with Roentgenographic Demonstration of Disintegration of Stone by Alkalinization, Surg. Gynec. & Obst. **38**:51-57 (Jan.) 1924.

So far as cystin lithiasis goes, the procedure outlined was theoretically and practically a success and leads us to hope that similar methods for dissolving more common varieties of calculi may be found.]

*Renal Tuberculosis.*—Braasch and Scholl [25] have found that repeated examinations of the urine for the bacillus of tuberculosis and guinea-pig inoculations at variable intervals may be the only method of establishing the diagnosis of renal tuberculosis. In patients with increased resistance, the usual urinary symptoms may be absent and the urine normal save for an occasional pus cell. When repeated guinea-pig inoculations with the urine from one kidney are positive, even though no other clinical data are present, the inoculation results may usually be regarded as sufficient to warrant operation. The renal lesion in such cases is characterized by encapsulation of the infected renal areas. The number of pus cells found on microscopic examination of the catheterized urine are not at all indicative of the extent of the lesion. The finding of a few pus cells in the catheterized urine of the supposedly healthy kidney is not of much practical significance. Bilateral tuberculosis occurs much more commonly than the usual clinical data indicate. In cases of bilateral renal tuberculosis in which only one kidney is markedly diseased, removal of this kidney is justified. Tuberculosis in the supposedly healthy kidney is probably a common cause of death within a year or two after nephrectomy.

Rydgaard [26] states that genital tuberculosis may arise from several sources: (1) Hematogenous infection, (2) the prostate, and (3) the upper urinary tract. He analyzed seventy-seven cases of true genital tuberculosis from Rovsing's clinic. In fifty-eight, the epididymis was diseased, the testis being free or almost free; in twenty-four, the condition was unilateral, and in nineteen, bilateral. In nineteen cases, it was necessary to remove the testis with the epididymis. Over 60 per cent. had no clinical signs of tuberculosis of other organs; 40 per cent. had tuberculosis elsewhere, either acute or in a quiescent stage. No relation between genital tuberculosis and gonococcal infection was noted. A number of patients gave a history of trauma shortly before the onset of epididymitis. The possibility of clean-cut healing was diminished in proportion to the duration of the disease. In from 50 to 75 per cent. of patients not operated on, the opposite epididymis became infected; with early operation, only six had involvement in the

25. Braasch, W. F., and Scholl, A. J.: Possible Errors in the Diagnosis of Renal Tuberculosis, J. A. M. A. **82**:688-692 (March) 1924.

26. Rydgaard, F.: Die Genital Tuberculose bei Mannern und die Resultate ihrer Behandlung unbesondere die Resultate der Epididymekitomie, Arch. f. klin. Chir. **123**:758, 1923.

opposite epididymis. In none did the disease recur in the testis, but four patients had recurrence in the urinary tract from three to ten years after epididymectomy.

[ED. NOTE.—Coincident tuberculosis in the genitalia and urinary tract rarely occurs in the female patient, but was noted in 171 (73 per cent.) of 234 male patients (Braasch). There is a high percentage of recoveries among patients who have clinical evidence of tuberculosis in the prostate and seminal vesicles which should contraindicate early surgical treatment of these organs. Spontaneous healing occurs in the majority of these cases. Tuberculosis in the epididymis should be treated surgically because of the frequency of suppuration and because the process does not usually become dormant and disappear. Furthermore, tuberculous epididymitis is always a possible source of a general infection.]

Ekehorn [27] observes that in most cases the dissemination of tuberculosis in the urinary tract is from above downward. This descending urinary infection is carried by the urine. Tuberculosis of the bladder heals when the original focus in the kidney is removed by nephrectomy. Ekehorn has had one case of tuberculous cystitis which was secondary to bilateral tuberculosis of the epididymus. In this case, the process in the bladder healed after the removal of the epididymides.

The kidney is infected with tuberculosis through the blood stream, which, however, does not take place through the process of elimination, but by direct hematogenous infection, due in most cases to infected emboli lodging in the kidney. It has been shown that emboli in the kidney usually affect the pyramids.

From the original focus a cavity develops, which is filled with tuberculous bacilli. This cavity later breaks through into the pelvis, with the discharge of many organisms into the urine. In this manner, secondary infection may now be spread by the urine to the renal pelves, ureters and bladder. Infection may also be carried by lymphatics from the original focus toward the cortex.

The author states that tuberculosis bacilli are most often and easily found in the early stages of the disease. He discusses the usual symptoms fully. In his discussion of the treatment of renal tuberculosis, he is of the opinion that nephrectomy is the only advisable procedure. Treatment by the roentgen ray, radium and tuberculin is not only unsatisfactory, but may be absolutely harmful. In bilateral cases, he advises removal of the worst kidney, if the disease is not far advanced in the other.

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27. Ekehorn, G.: The Diagnosis and Treatment of Tuberculosis of the Kidneys and the Urinary Tract, Tubercle, London **4**:337-345 (May) 1923; **4**:393-398 (June) 1923; **4**:444-449 (July) 1923.

Judd and Scholl [28] report the results following surgical treatment for renal tuberculosis of 874 patients. Nephrectomy was performed on 863 and exploration only on nine. Complete postoperative data were obtainable concerning 611 patients. Usually, complete lumbar nephrectomy was performed. In a few instances, transperitoneal nephrectomy was performed or the peritoneal cavity was opened while the kidney was being removed by the lumbar route; such contamination of the peritoneal cavity markedly increases the operative risk.

In the presence of a perinephritic abscess, removal of the kidney and drainage of the abscess at the same time increases the operative risk. Two of eight patients died following such procedures. None of the eight died on whom the two-stage operation was performed. In eighteen cases of bilateral infection, one kidney was removed. Four patients died from anuria immediately after operation and ten during the next eighteen months. Twenty-three patients (2.7 per cent. of 845 who had had unilateral nephrectomy) died the first month after operation. One hundred and ninety-one (31.2 per cent. of the 611 patients) are dead; 358 (58.6 per cent.) are completely cured on an average of four years after operation, and sixty-two (10.1 per cent.) are still having urinary trouble.

Hunt [29] discusses the surgical treatment of tuberculosis of the genito-urinary tract. Primary genito-urinary tuberculosis probably never occurs, tuberculosis being demonstrable elsewhere in the body in approximately 80 per cent. of the cases. As regards the primary seat of tuberculosis in the genito-urinary tract, the theories of Barney that the epididymis is first involved as a result of hematogenous infection, and that of Young that the seminal vesicles are the primary site and the epididymis a secondary one, are discussed, the author holding to the former view.

The kidney is most often affected and the disease is usually unilateral. When unilateral tuberculosis of the kidney is present, nephrectomy by the lumbar route is indicated. In bilateral renal disease, nephrectomy is contraindicated unless there are acute unilateral complications. The association of acute tubercular processes elsewhere in the body must be taken into consideration. However, with the recent advent of local anesthetics and ethylene, even in the presence of progressive pulmonary tuberculosis, the risk for nephrectomy in cases of complicated closed renal tuberculosis may be considered minimal. Miliary tuberculosis may be taken as a contraindication for operation.

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28. Judd, E. S., and Scholl, A. J.: Surgery of Renal Tuberculosis, Ann. Surg. **79**:395-409 (March) 1924.

29. Hunt, V. C.: The Surgical Treatment of Tuberculosis of the Genito-Urinary Tract, Including the Kidneys, Bladder and Seminal Vesicles, Arch. Surg. **8**:811-819 (May) 1924.

The mortality rate for unilateral tuberculosis, if nephrectomy is done, is 2 per cent., and Braasch reports a later mortality of 20 per cent. within five years after nephrectomy, with a prognosis of partial recovery for 80 per cent. of the patients.

Persistent sinus after operation is due to incomplete removal of the diseased tissue, persisting infection in the ureter, and the institution of drainage. In extensive renal tuberculosis, the perirenal fat should be removed as completely as possible. Ligation and sterilization of the cut end of the ureter seems the method of choice, yet Judd's method of threading the cut end of the ureter into a rubber tube, closed at one end, avoids general contamination of the wound and is a satisfactory procedure. Low stricture of the ureter is best treated by ureterectomy. The insertion of drainage tubes is inadvisable.

Tuberculosis of the bladder is secondary to renal tuberculosis and occurs in about 75 per cent. of the cases. According to Braasch, 48 per cent. of those who have preoperative symptoms of less than three or four months' duration improve immediately after nephrectomy, and ultimate improvement may be expected in 60 per cent. Palliative suprapubic cystostomy is occasionally necessary in the protracted cases of tuberculosis cystitis of long duration. Especially is this true if the bladder is contracted and irritability and frequency are marked.

Tuberculosis of the seminal tract is often associated with renal tuberculosis, but the evidence that this is a hematogenous infection is emphasized. There is a tendency for epididymitis to become bilateral. Tuberculosis of the seminal tract usually occurs in the third and fourth decades. Associated tuberculosis elsewhere is common. It is usually considered that the testis becomes involved secondarily from the epididymis.

Hunt believes the conservative procedure of epididymectomy done is more desirable than the radical dissection of the epididymis, vas, vesicles, and prostate, when diseased. After epididymectomy, the process in the remaining portion of the seminal tract tends to subside. He recommends the high inguinal incision, even in cases with one or more scrotal sinuses. The contents of the scrotum are readily drawn up into the incision, and excision of the epididymis, the involved tunica vaginalis and sinus, together with the vas, as high as the inguinal ring, is thus facilitated. Castration is sometimes necessary, but if there is only partial involvement, the procedure of partial resection of the testis is recommended.

*Actinomycosis.*—A case of primary actinomycosis in the left kidney of a woman of advanced age is reported by Abbott.[30] Tumor in the

30. Abbott, D. P.: Primary Actinomycosis of the Kidney, J. A. M. A. **82:** 1414-1415 (May 3) 1924.

left side, pyuria, and fever with emaciation were prominent symptoms. Cystoscopic examination with pyelography gave the impression of hypernephroma, and operation was undertaken. The kidney mass was adherent to the vertebral column and the actinomycotic nature of the disease was recognized. The wound was partially closed, part of the kidney being left exposed for roentgen therapy. The patient died several months later after a steady decline. Necropsy revealed actinomycosis of the left kidney, with a lumbar abscess and destruction in part of the fourth and fifth lumbar vertebrae. Secondary nodules were found in the lungs, but these were so small as compared with the renal pathology that the author believes the condition was primary in the kidneys.

*Anomalies.*—Day [31] reports several rare anomalies of the kidney and ureter. One patient, a man aged 32, had a single meatus and ureter on the right side. The ureter crossed to the left at the sacroiliac juncture with the calices facing to the right, as if the kidney had been normally placed on the right side instead of on the left, just above the iliac crest.

A man, aged 21, had an ectopic opening of the left ureter into the posterior urethra and pyonephrosis of the left kidney. A left ureteronephrectomy was performed. A perineal operation was then undertaken to drain the sac and to dissect out the terminal portion of the ureter coursing through the base of the left lobe of the prostate.

A woman, aged 34, had pain in the back, a fluctuant tumor in the left renal region, moderate pyuria and bacteruria, and multiple shadows in the left kidney area, extending from above the twelfth rib to 2 cm. below the iliac crest. The left ureter was injected, the opaque mediums extending only to the fifth lumbar vertebra. At operation, the left kidney was removed; its substance was thinned out, and the pelvis contained an enormous number of multiple calculi, resembling bird seed. The ureter was absent. Day explains the situation as due to stenosis and angulation at the ureteropelvic juncture, with necrosis and separation of the ureter which retracted downward towards the bladder.

A patient, aged 21, died of tuberculosis. At necropsy, the left ureter did not terminate in a meatus as it pierced the bladder wall, but extended through the wall of the bladder, made a loop across the trigon and became adherent through a thick, fibrous cord to the right of the trigon, extending to the internal lip of the right meatus. The lumen of this tube extended fully to the median line of the bladder and appeared to have been patent all the way across to the right and to have been continuous with the right ureter.

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31. Day, R. V.: Some Rare Anomalies of the Kidney and Ureter with Case Reports, Surg., Gynec. & Obst. **38**:51-57 (Jan.) 1924.

Four cases of congenital absence of one kidney are reported: One case with a normal ureteral meatus, but no ureter on the side of the absent kidney; one with no ureter or meatus on the anomalous side; one with the ureter extending to near the normal kidney position, but no kidney tissue or evidence of renal vessels in this area, and one in which the single kidney was normally placed, with complete duplication of pelvis and ureter. Cystoscopically, the right and left ureteral orifices appeared normally placed in the bladder.

*Cysts.*—Two cases of solitary large serous cyst of the kidney are reported by Harpster, Brown and Delcher.[32] Nephrectomy was performed in both cases. One case was complicated by cholecystitis and gallstones, and the patient died four days after operation. The other recovered.

The authors analyze ninety-five cases collected from the literature. The upper pole of the kidney is most frequently involved, but the cysts may arise at any point. In one case, the cyst contained 12 liters of fluid. Tumor and pain from pressure are the chief clinical features of these cysts. Hematuria is present in less than 10 per cent. of the cases.

Diagnosis preoperatively is difficult, hydronephrosis, cysts of the liver, spleen, pancreas, gallbladder, ovary and mesentery giving at times similar symptoms.

The authors feel that the pathogenesis is probably an intense interstitial nephritis, with an associated obliterating endarteritis, but whether this has an embryonal or a later origin is dubious. The treatment recommended is excision of the cysts, including any part of the kidney involved, or total nephrectomy, if the kidney is badly diseased or if the cyst is difficult of enucleation. In the cases reported, a 33 per cent. mortality followed nephrectomy; 10 per cent. in the cases in which the cyst was excised, and 50 per cent. in cases in which the cyst was tapped. Partial nephrectomy seems to be relatively free from the danger of hemorrhage, if proper suturing is done.

### URETER

*Carcinoma.*—Kretschmer[33] collected thirty-four cases of primary carcinoma of the ureter and reports one of his own. The patient was a man, aged 74. Hematuria was the initial symptom. A right pyelogram showed that the ureter went upward and outward in an S-shaped curve, the tip overlying the middle of the crest of the ilium. At the level of

32. Harpster, C. M.; Brown, T. H., and Deleher, A.: Solitary Unilateral Large Serous Cysts of the Kidney, with Report of Two Cases and a Review of the Literature, J. Urology **11**:157-175 (Feb.) 1924.

33. Kretschmer, H. L.: Primary Carcinoma of the Ureter, Surg., Gynec. & Obst. **38**:47-51 (Jan.) 1924.

the second lumbar vertebra, a dense rounded shadow, 3 cm. in diameter, was seen. A right lumbar nephro-ureterectomy was performed, and the patient made an uneventful recovery.

The location of the growth is commonly the lower ureter. However, any portion may be involved. Among the symptoms, hematuria is most prominent, being present in twenty-four of the thirty-five cases reported. Pain was constant in twenty-six cases. Diagnosis can be made on physical examination, by abdominal palpation, by vaginal or rectal palpation, by cystoscopy, ureteral catheterization and pyelography. Persistent bleeding after ureterectomy is significant of neoplasm and indicates incomplete resection or recurrence in the stump. The patient in the case reported had such a history. The author quotes the preoperative diagnoses that were made by different observers, and the variety shows the difficulty with which the condition is to be differentiated from other diseases, particularly from renal neoplasm and hydronephrosis, and papilloma of the kidney with extension into the ureter.

### BLADDER

*Diverticulum.*—Swift Joly [34] reports fourteen operative cases of vesical diverticula. He disapproves of preliminary drainage of the bladder when it can be avoided, but when necessary he advises placing two small tubes in the diverticulum besides draining the bladder. He points out that no single type of operation is suited to all cases and discusses three types of operation which are most useful. Excision of the diverticulum from without the bladder is suitable for diverticula high up on the lateral walls or at the urachus. This operation may be performed either extraperitoneally or transperitoneally. The bladder may be opened and the diverticulum inverted and removed through the bladder, or the bladder wall may be split down to the orifice of the diverticulum and the sac incised.

When hypertrophy of the prostate is associated with the diverticulum, Joly removes both at one operation, if possible. When this is impossible, the diverticulum should be attended to first and the prostate later. Lithopaxy should not be attempted where vesical stone is associated with diverticulum.

Blum [35] believes that there is a congenital predisposition to the formation of diverticula in certain parts of the bladder. Acquired mechanical or nervous obstruction to the urinary outflow is only the

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34. Joly, J. S.: The Operative Treatment of Vesical Diverticular, Lancet **2**:445-452 (Sept. 1) 1923.

35. Blum, V.: Drei neue Falle von Erfolgreicher Operation von Harnblasen divertikeln, Ztschr. f. urol. Chir. **5**:90-100 (July) 1920; Diverticulum in the Bladder, Ztschr. f. urol. Chir. **12**:290-302 (March) 1923.

activating cause which brings the latent defect into prominence and causes gradually increasing dilatation. In two articles he reports twenty cases of bladder diverticula. In eight cases, transvesical extraperitoneal excisions were performed with good results; there were no postoperative deaths. In two of these, the prostate was also removed. In one case, a transvesical removal with transplantation of the ureter was performed. This patient died six months after operation. An extravesical extraperitoneal operation was performed in two cases. There were no deaths, and good results were obtained in both cases. A transperitoneal extravesical excision was performed in two cases; one patient died in twenty-four hours. In one case, a prostatectomy and obliteration of the diverticular sac was performed; in a second case, the dome of the bladder was removed, and in a third, intra and extraperitoneal excision. Good results were obtained in all cases. In one case, the neck of the sac was dilated. One patient died following dilatation of the sac and excision of the internal urethral sphincter. One case was complicated by an extensive hemangioma of the wall of the diverticulum; this caused marked hemorrhage. In a second case, a vesico-intestinal fistula complicated the diverticulum. In both cases, the wound healed completely. In eight cases, the patients are perfectly well, except that one has a hernia in the scar. Three patients have a persistent cystitis; one has a recurrence of the diverticulum; four are still under observation, and four are dead. One died six months after operation from bilateral pyelonephritis; one died two months after operation from bilateral renal stones; a second patient died of shock following operation; a third died from uremia.

Judd and Scholl [36] report 133 cases of diverticulum of the bladder. These cases were studied with regard to the type of operation performed and the postoperative results. One hundred and thirty-one of the patients were men and two were women. Complete postoperative data were obtainable in 110 (83.9 per cent.). In thirty-two per cent. the bladder contained multiple diverticula; these occur most commonly at the prostatic age. Sixty-nine and two-tenths per cent. of the patients were between 50 and 70 years of age. The most common location of the orifice is the region of the ureteral outlet; 87 per cent. of ninety single diverticula were found in this location. The diverticula were completely excised in fifty cases. Three patients (6 per cent.) died. In thirty-seven cases in which there was obstruction of the vesical outlet the diverticulum was excised and prostatectomy performed. Three patients died (8.1 per cent.). In forty-six cases, the diverticulum was not removed. The ureter was involved in the diverticulum in five

36. Judd, E. S., and Scholl, A. J.: Diverticulum of the Urinary Bladder, Surg., Gynec. & Obst. **38**:14-26 (Jan.) 1924.

cases; in one it was transplanted to a healthy portion of the bladder. In four cases in which the ureter was dilated, it was ligated, cut, and allowed to drop back into the wound. In twenty cases, the diverticula were associated with stones. In nine cases, the calculi were in the diverticula, in eight in the bladder, and in three, both in the bladder and the diverticulum. Carcinoma occurred in a diverticulum in four cases; three of the patients died shortly after operation, and the fourth died two years after resection of the diverticulum. Six patients had carcinoma of the bladder and diverticulum. Three of these patients died shortly after operation; one lived sixteen months, and one three years; the sixth had a recurrence eight months after excision of the involved area. Of the six patients who died following excision of the diverticula, five died of renal infection and obstruction. In most instances they had both acute and chronic nephritis. The sixth patient died of pulmonary embolism; this patient also had chronic nephritis.

*Tumors.*—Kidd [37] reviews the results of treatment in his series of 162 cases. He observes that the cause of death was not so much the spread of the disease to distant parts as to local accident, such as exhaustion from pain and bleeding and interference with ureteral drainage leading to dilated ureters, pyelitis and uremia. Tumors of the bladder break through the muscular coat rather late, but may spread along the submucous layer, giving rise to extension throughout. Hematuria is often a neglected symptom and much valuable time is lost while palliative internal medication is being used. He disapproves of taking specimens of bladder tumors through the cystoscope for pathologic examination and prefers to trust to cystoscopic appearances and to the surgeon's judgment. Of twenty-eight patients with simple papillomas or small recurrent tumors, noted after operation and treated by diathermy, twenty-one have been traced. Of these, nineteen have no further recurrence, fifteen having been well for more than four years. Only two have recurrences. Two patients with inflammatory papillomas were treated satisfactorily with diathermy. Twenty-six patients had malignant papillomas or early carcinomas; the condition of twelve was resistant to diathermy; in the remainder the results were good. Twenty-eight patients had partial cystectomies, and thirteen were cured. Seventeen patients had cystotomies with local removal of the tumor, and nine were possibly cured, while five were positively cured. These results were not so favorable as those following partial cystectomy. Five patients, who had total cystectomy, did not survive over three weeks.

Radium treatment is not considered sufficiently reliable to replace surgery, except when total cystotomy would be the alternative.

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37. Kidd, F.: The Treatment of Epithelial Tumors of the Urinary Bladder, Lancet **1**:523-527 (March 17) 1923; **1**:582-586 (March 24) 1923; **1**:636-639 (March 31) 1923.

Young and Scott [38] review 380 cases of bladder tumor treated at the Brady Institute. The majority were located in the trigon and around the ureteral orifices. The anterior wall is less often affected, and the vertex and upper lateral walls rarely are the site of tumors. Resection with the removal of a wide margin of bladder wall gives good results in the vertex, anterior and upper lateral walls; however, good results also follow resection in the trigon and around the uretral orifices. There were 27 per cent. of five-year cures in fifty-one cases in which the wall of the bladder was resected. Deep cauterization is recommended if the vesical neck and prostate are involved. Fulguration is advised for benign papillomas, and fulguration and radium combined for the larger malignant growths. Although the results of resection are good, less radical procedures are indicated if the growth is extensive, as large resections carry a high mortality. By the judicious use of fulguration, radium, the electrocautery and careful radical resection, 95 per cent. of the benign, 75 per cent. of the malignant papillomas, and 25 per cent. of the infiltrating carcinomas are probably curable.

Lavenant [39] describes a case of colloid carcinoma of the dome of the bladder, which was excised under spinal anesthesia. The peritoneal covering of the bladder was involved; in order to remove the tumor it was necessary to open into the abdominal cavity. The peritoneum was opened and sutured to the bladder below the level of the growth before the bladder was opened. The upper half of the bladder was removed. On histologic examination, the growth was found to be a glandular carcinoma similar to intestinal carcinoma. The tumor was of the same type as growths described by Albarran and Hartman as being tumors of allantoic derivation. It was only of a moderate degree of malignancy; two years later there had been no recurrence.

[ED. NOTE.—Glandular tumors occasionally are found in the bladder, and sometimes are noticed undergoing colloid degeneration. These tumors, differing from all other vesical neoplasm, have a predilection for the upper areas of the bladder, particularly the dome, which suggests an origin from remnants of embryonal inclusions. DeKorte has described a glandular type of carcinoma removed from the apex of the bladder in a woman, aged 50. Adami, who examined this tumor, concluded that it originated from urachal remains in the wall of the bladder. Barringer reported an adenocarcinoma involving only the apex of the bladder; the larger portion was above the bladder. Ewing, who examined the tumor, said that the growth was unique, and quite

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38. Young, H. H., and Scott, W. W.: The Results Obtained by Various Methods in the Treatment of Tumors of the Bladder, New York M. J. **118**: 262-269 (Sept. 5) 1923.

39. Lavenant, A.: Epithelioma Colloide d'origine allantoidienne due sommet de la vessie, Jour. d'urol. med. et chir. **17**:43-46, 1924.

possibly originated from a remnant of the allantoic end of the bladder, or possibly developed from a cloacal inclusion. Such tumors are only moderately malignant. They grow slowly, and when in the dome cause few symptoms. They extend in the perivesical tissues readily and have a marked tendency to recur.]

### PROSTATE

Lichtenstern [40] performs his prostatectomies under local anesthesia. The neck of the bladder and the suprapubic tissues are injected with a long needle inserted in the suprapubic region. Two hundred cubic centimeters of a 1 per cent. solution of eucain hydrochlorid is left in the bladder for thirty minutes before the operation is started. In most cases, a one-stage suprapubic operation is performed. In infected cases in which there is chronic urinary retention, and the bladder will not tolerate a permanent catheter, the operation is performed in two stages. After enucleation of the prostate, the bleeding is controlled with catgut sutures and the capsule packed. The bladder and abdominal wall are carefully closed about the drainage. The packing is removed from the capsule on the third or fourth day and the drainage tube on the eighth. Closure of the wound is facilitated by a urethral catheter. In cases in which infection is marked, the bladder is lavaged and intravenous injections of hexemethylenamin given.

Thomson Walker [41] emphasizes the value of careful preoperative treatment in avoiding postoperative complications after prostatectomy. He believes that two elements are of great importance in bowel complications; one is flatulent distention due to intestinal fermentation, and the other is exhaustion of the sympathetic nervous system due to uremia, intestinal toxemia and shock. These should be combated by preoperative treatment.

Complications of the kidney should usually be anticipated and treated before operation. Careful study of the renal function is imperative. The author advised estimation of urea concentration in the urine, in conjunction with the estimation of blood urea. He does not approve of correlating these in the form of a factor or index. Early operation is recommended, and as the proportion of early operations increases, the necessity of two-stage operations decreases. By doing the one-stage operation, a better controlled operation can be performed. In 100 consecutive cases he reports malignant changes in sixteen, and doubtful or precancerous change in fourteen. In most instances, there was no

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40. Lichtenstern, K.: Weiner Urologische Gesellschaft. Ztschr. f. urol. Chir. **14**:100-101, 1923.

41. Walker, J. W. T.: Some Problems of Prostatectomy, Brit. M. J. **1**:133-137 (Jan. 27) 1923.

suspicion of malignant change before operation. Sepsis is usually avoidable by suitable preoperative drainage of the bladder. Sufficient drainage after operation is also a factor in avoiding infection. Spinal anesthesia is considered the anesthetic of choice, although gas and oxygen has few disadvantages and is very useful in the single-stage operation when relaxation is not so essential.

Hinman [42] believes that the popularity of the suprapubic method of prostatectomy is due to a conviction that the suprapubic removal is technically easier, and the functional result therefore more certain to be satisfactory because of the more certain absence of any surgical defect. The technical difficulties of perineal removal account for the general disfavor in which it is held. However, when the operation is performed successfully and correctly, as good or better functional results are obtained as by the suprapubic method. The perineal route is recognized as being the more benign of the two methods and should be the method of choice for this one reason. The technical difficulties of the operation consist primarily in keeping out of the rectum, in preserving the external sphincter from injury as a direct effect of surgery, or as an indirect one of adhesions or scar formation, in protecting the ejaculatory ducts from destruction and occlusion and in securing a clean, complete removal of the obstructing prostate in such a manner as to prevent recurrence and at the same time avoid structural defect.

Caulk and Sanford [43] analyze one hundred cases of the bar type of obstruction of the vesical neck and the collar contracture type. The collar type varies from slight ingrowths of the sphincteric region to dense scleroses which are true contractures of the vesical neck.

Ninety cases of the one hundred were selected as suitable for the authors' cautery punch operation. Symptoms common to obstruction from other causes are noted: Nocturia, frequency, urgency, and retention. Following the punch operation, these subsided to a great degree in the majority of cases. There was no incontinence. In the one hundred cases, cystoscopy revealed thirty-six bars, twenty-seven obstructions of the collar type, fourteen definite contracted necks, four lateral lobules, and seven cancers. The technic of the cautery punch operation is described. The alternating current is advised in the use of the cautery rather than the direct current. Hemorrhage is rare. Most of the patients leave the hospital after two days. Epididymitis occurred in seven patients. The end results were good, there being immediate

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42. Hinman, T.: The Standardization of Prostatectomy with Reference to the Recent Modification of Young's Technic, Surg. Clin. N. America **3**:717-730, 1923.

43. Caulk, J. R., and Sanford, J. H.: An Analytical Study of 100 Cases of Selected Vesical Neck Obstruction Operated on by the Author's Cautery Punch, J. Urology **11**:45-63 (Jan.) 1924.

improvement of the stream and reduction of the residual urine. In eight cases of inoperable cancer with obstruction, the punch was used with satisfactory results seven times.

Liebig [44] presents a collected review of various types of prostatectomy with indications for the different routes of removal and the results obtained. These are divided into two sections, from 1912 to 1916, and from 1917 to 1922.

*Types of Prostatectomy*

| | Number | Deaths | Per Cent. |
|---|---|---|---|
| Perineal removal | | | |
| 1912 to 1916 | 279 | 29 | 10.0 |
| 1917 to 1922 | 759 | 22 | 2.9 |
| | 1,038 | 51 | 4.9 |
| Ischiorectal (Voelcker type) | | | |
| 1912 to 1916 | 407 | 21 | 5.1 |
| Suprapubic (single stage) | | | |
| 1912 to 1916 | 2,958 | 248 | 8.38 |
| 1917 to 1922 | 2,379 | 189 | 7.9 |
| | 5,337 | 437 | 8.1 |
| Suprapubic (two-stage) | | | |
| 1912 to 1916 | 276 | 28 | 10.2 |
| 1917 to 1922 | 507 | 22 | 4.3 |
| | 783 | 50 | 6.3 |
| Total suprapubic operations | 6,120 | 487 | 7.9 |
| Grand total | 7,565 | 559 | 7.4 |

[ED. NOTE.—These statistics indicate a large increase in the number of perineal prostatectomies performed in the last five years and a corresponding decrease in the mortality following operation, the total mortality being lower than that following suprapubic prostatectomy. A large number of the perineal prostatectomies were performed by urologic surgeons of unquestioned skill, while the suprapubic method is the one selected by the general surgeon.

The statistics on Voelcker's ischiorectal type of prostatectomy are from the work of only four surgeons, and the results were estimated during the formative stage of the operation. The percentage of two-stage suprapubic operations has greatly increased during the last five years and is attended by a much lower operative mortality.]

Foss [45] describes a gauze pack consisting of accordion pleated gauze traversed by tape. The distal end of the tape is drawn through the urethra by a previously inserted catheter; tension on the tape draws the folds of gauze into the prostatic fossa, the proximal end of the tape being held by a button which presses on the pack and holds it in place. The distal or outside end of the tape is fastened snugly against the glans penis. A second tape is sewed to the proximal end of the pack and

44. Liebig, F.: Die Prostata hypertrophie, Ztschr. f. Urol. **17**:593-613, 1923; **17**:626-659, 1923.

45. Foss, H. L.: The Control of Hemorrhage Following Prostatectomy, Ann. Surg. **78**:802-805 (Dec.) 1923.

comes out through the suprapubic wound. This tape is used to withdraw the pack when the portion of the first tape attached to the penis is released.

[ED. NOTE.—Control of prostatic hemorrhage remains a technical problem of importance. The Hagner bag, the simple gauze pack, and suture of the prostatic capsule alone each has its advocates, who are able to employ their individual methods successfully. Whether Foss' device will prevent the occasional hemorrhage that one sees with various methods is a matter for clinical experience to prove.]

In a series of twenty-eight cases of prostatic enlargement, Swan [46] found four in which carcinomatous areas were found on histologic examination. In none of these cases was there obvious or even suspicious gross changes indicating carcinoma. He quotes Estes and Fletcher as finding 174 (25.7 per cent.) cancerous and fifty-eight (8.6 per cent.) borderline or precancerous prostates in the 648 cases of prostatic enlargement submitted to them for pathologic examination. Wade found 10.4 per cent. cancerous prostates in 134 removed as clinically benign, and J. Thomson Walker sixteen in one hundred cases.

Walker [47] describes the operation of transpubic prostatectomy for carcinoma with subsequent restoration of the urethrovesical attachment. An incision is made from one inch below the umbilicus to the symphysis, the bladder is opened, the cartilage joining the pubis is divided, the bones separated, and the attachments of the pubic and triangular ligaments divided. The pubis is separated by a special retractor and the prostate exposed. By blunt finger dissection the prostate is freed from the surrounding tissue. The urethra is cut across at its juncture with the prostate. The prostatic end of the urethra is pulled upward and the prostate dissected free from the rectum. This is carried up not quite to the bladder, which is then divided transversely as near the vesical neck as is warranted by the growth. The prostate and divided end of the vesical neck is removed. Care is taken to protect the internal sphincter. The end of the urethra is sutured to the posterior vesical margin, and a catheter passed. The suture of the bladder and urethra around the catheter is completed, followed by closure of the bladder from its juncture with the urethra upward, leaving an opening above for the insertion of a drainage tube. As closure of the symphyseal division with silver wire failed to hold, a snugly fitting binder to hold the pubes together is recommended. A drain is placed in the bladder and the wound closed.

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46. Swan, R. H. J.: The Incidence of Malignant Disease in the Apparently Benign Enlargement of the Prostate, Lancet **2**:971-974 (Nov. 3) 1923.

47. Walker, G.: Transpubic Removal of the Prostate for Carcinoma, Ann. Surg. **78**:795-801 (Dec.) 1923.

The patient is put on a Bradford frame and kept immobile for one week, daily irrigation being carried out with saline after the second day. The catheter is kept in place for two weeks. After removal of the catheter a sound is passed every third or fourth day until the wound is healed. A back rest is allowed at the end of two weeks, the patient being allowed out of bed at the end of the third week.

Two cases are described in which the operation was performed satisfactorily; in one of these there was recurrence.

[ED. NOTE.—Cancer of the prostate remains a problem both for the surgeon and for the radiotherapist. The operation described removes only the local growth; no account is taken of regional lymphatic spread or distant metastasis. The prognostic importance of metastasis has been shown in dealing with cancer elsewhere, and the work of Bumpus indicates that these factors are of prime importance in the outlook with regard to prostatic cancer.

The question arises as to whether the end result of a radical operation, such as is described by Walker, will be of any greater value than radium treatment, or enucleation of obstructive portions of prostatic cancer in conjunction with radium treatment.]

### URETHRA

Montalègre [48] collected thirty-nine cases of congenital stricture of the posterior urethra. He divided such cases into (1) a diaphragm, either partial or complete, perpendicular to the long axis of the urethra; this is similar to the condition Bazy describes as a persistence of the cloacal membrane, and (2) one or two valves spreading out from the verumontanum. These valves may be fused and cause almost complete urethral obstruction, or there may be but a single large valve. Thompson compared these valves to an imperforate hymen. In the removal of these obstructions, they may be destroyed directly by forcing a sound through the urethra, by a punch operation, or by fulguration. In cases in which the obstruction is high in the urethra, a suprapubic exposure and retrograde destruction of the obstructing mass is desirable. Heresco, Poppert, and others have described a perineal approach through an incision in the posterior urethra for the removal of the obstruction.

Ward [49] describes a patient with an extensive vesicovaginal fistula caused by trauma during the delivery of a full term fetus by craniotomy. The opening was at the site of the internal meatus and involved the

48. Montalègre, M.: Obstruction et retracessements congenitaux de l'uretre posterior, Jour. d'urol. med. et chir. **17**:160, 1924.

49. Ward, G. G.: Reconstruction of the Urethra After Complete Loss Complicating an Extensive Vesicovaginal Fistula; the Operative Technic Employed with Success, Surg., Gynec. & Obst. **37**:678 (Nov.) 1923.

loss of the anterior vaginal wall, the floor of the urethra, and a portion of the trigon. Before coming under Ward's care, the patient had had an exploratory laparotomy with incision into the uterus and passage of a sound and catheter through the uterine canal. This had established a passage from the uterus to the vagina. Ward, during the first operation, attempted to close the fistula by making incisions through the scar tissue of the vaginal vault to the suprapubic arch and above, and on either side of the urethral site. The edges were dissected free from the bladder wall for about 1 cm., and the bladder opening closed with interrupted catgut sutures. The vaginal edges were united with silkworm gut. A soft catheter was inserted. At a second operation, a small fistula was found. The edges were freshened and interrupted sutures were passed to narrow the orifice in the hope of giving vesical control. The result was partly satisfactory, but leakage in the standing posture continued. A third operation was performed and reconstruction of the urethra, of which the superior wall alone remained, was attempted. The edges of this wall were undermined, and the loosened lateral wall sutured inferiorly around a catheter passed into the bladder. The cut edges of the vaginal wall were then brought together over the new urethra, and sutured with silkworm gut sutures. Failure resulted from sloughing of the tissues. A fourth attempt was successful. Kelly's technic of making a tunnel under the vestibule and drawing through it a long flap, dissected from the anterior vaginal wall with its end left attached to the vesical opening, was carried out. This left a thin layer of tissue posteriorly and the bladder wall exposed in such a way that it could hardly heal by granulation. Accordingly, Noble's technic of closing over this denuded area by a pedicle flap from the right labium minoris was employed. A retention catheter was placed in the new urethra and satisfactory healing and vesical control followed.

[ED. NOTE.—Vesicovaginal fistula, is in itself, a troublesome condition, and when complicated by the urethral destruction, presents a formidable surgical problem. Plastic repair means painstaking application of surgical ingenuity of the highest type. The early work of Emmet and Kelly has established principles of using adjacent mucous membranes from the vaginal wall, of tightening the tissues surrounding the internal sphincter, and most of all, of persistent repetition of effort in the face of failure.]

Unger and Höring [50] describe a method of treating urinary incontinence in the female. The lower rectus fascia is exposed through a suprapubic transverse (Pfannenstiel) abdominal incision. A tongue of tissue 8 cm. long by 4 cm. wide, with the base at the pubes, is cut

50. Unger, E., and Höring, F.: Zur Behandlung der Incontinentia urinae, Ztschr. f. Urolog. Chir. **12**:96-99 (March) 1923.

from the fascia and underlying rectus muscles. This is split longitudinally. The neck of the bladder is then freely exposed, and the urethra, which contains a catheter, is partially isolated from the surrounding tissue. The perineal region and vaginal wall are then incised from below, and a communication made between the vagina and the suprapubic incision. The tongue of muscle and fascia is brought down; the urethra being fitted in the longitudinal split. The ends of the fascia which are not under tension are sutured beneath the urethra. These ends are crossed, but not tied or doubled. The vaginal wound is closed. The defect in the rectus muscle is closed over by approximating the cut muscles and fascia. A permanent catheter is left in place for several days. In most cases in which this has been carried out, satisfactory results were obtained. Stoeckel, who used this method in twelve cases, had satisfactory late results in ten cases.

[ED. NOTE.—Urinary incontinence is a fairly common and distressing feature in women. The simple urethral plastic operation, as devised by Kelly, is sufficient in most instances, but if patients do not respond to minor plastic methods, this rather extensive procedure might be indicated.]

Garlock [51] describes the repair of a ruptured urethra and hematoma which followed indirect violence. A median-line perineal incision was made over the hematoma, and a sound passed through the anterior urethra. It was impossible to find the posterior or proximal portions of the urethra. Cystotomy was performed, and a retrograde passage of a sound brought the proximal portion of the urethra into view; the two ends were sutured over a catheter. Drainage of the bladder through a suprapubic wound and through a urethral catheter was carried out for eleven days. Urethral sounds were passed daily following the removal of the catheter. Seven months later, good results had been obtained.

### TESTIS AND SEMINAL VESICLES

Southam and Linell [52] report thirty-eight cases from the Manchester Royal Infirmary and seven cases from other sources, in which operation was performed for neoplasm of the testes. In seven of the cases, the testes were undescended. Trauma appears to precede malignant changes in certain cases. The malignant tumors are classified as (1) carcinomas: spheroidal cell, spermatocytoma, and chorioepithelioma, and (2) sarcoma.

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51. Garlock, J. H.: Rupture of the Urethra, Surg., Gynec. & Obst. **37**:533-537 (Oct.) 1923.

52. Southam, A. H., and Linell, E. A.: The Pathology of Neoplasms of the Testis, Brit. J. Surg. **11**:223-233 (Oct.) 1923.

The spermatocytoma is the most common type; the average age of patients affected is forty-two and one-half years. The true spheroidal celled carcinoma is less common, and occurs earlier; the average age of patients is twenty-six and three-tenths years. The tumors apparently arise from malignant metaplasia in the epiblastic and hypoblastic cell elements in a teratoma.

The benign tumors are classified as teratomas and mixed cell tumors, and are liable to malignant change. All tumors of the testes are potentially malignant, and should be removed surgically.

Hinman, Gibson, and Kutzman [53] review the history of operations for teratoma testis, showing that castration alone is of relatively little value. Removal of the lymphatics, together with the testicular tumor and as much of the vas as possible, gives much more satisfactory results. Seventy-nine cases of testicular tumors were studied, and it was found that the time the growth has existed has little bearing on lymphatic involvement. The teratoma (mixed-cell tumor) spreads more rapidly than the seminoma (unicellular tumor). Castration in the cases of teratoma offered a poorer prognosis than was obtained following operation for the seminomas. Castration alone will cure only from 15 to 20 per cent. of malignant tumors of the testis. The operation must antedate glandular metastasis in order to obtain a satisfactory result. A radical operation was performed in all cases in this series. Nine patients had palpable metastatic glands; all died shortly after operation. Thirteen patients had inoperable metastatic glands, not palpable clinically, at operation; consequently, in twenty-two of seventy-nine the condition proved to be primarily inoperable. Of twenty-five patients without glandular metastasis at operation, eight died later of metastasis, and only eight remained living and well. Twenty-six patients from whom the glands were removed at operation, were malignant; seventeen are now living and well. In all, 35 per cent. of the seventy-nine patients studied, had been cured by radical operation, in contrast to the usual 15 per cent. cured by castration alone.

[ED. NOTE.—The removal of regional lymphatics in cancer of the breast, lower lip, and cancers of the stomach, has been demonstrated to be a great factor in the control of malignant disease. Block dissection has thus been extended into a new field. In the old Wertheim operation for uterine cancer, removal of the regional lymphatics was attempted in a manner comparable to the technic outlined by Hinman and his associates. This feature of the Wertheim operation has fallen into disuse because of the prolonged operative procedures and consequent mortality. The immediate operative mortality of the seventy-nine

53. Hinman, F.; Gibson, T. G., and Kutzman, A.: The Radical Operation for Teratoma Testis, Surg., Gynec. & Obst. **37**:429-451 (Oct.) 1923.

radical operations for teratoma testis was only 12.5 per cent., so that this form of treatment appears to be worthy of further clinical trial as a method of conquering a disease which, with simple castration, will only yield about 15 per cent. of cures.]

A fibroma of the spermatic cord, a proliferating epithelial tumor of the epididymis, which was considered of dubious malignancy by several pathologists, and a seminoma of the epididymis are reported by Hinman and Gibson.[54]

Tumors of the spermatic cord comprise 90 per cent. of the epididymis, spermatic cord, and testicular tunics. In order of frequency of occurrence, the types of tumors of the cord are lipomas, sarcomas, dermoids, fibromas and myxomas. It is emphasized that lipomas must be differentiated from fat hernia which is much more common, and which may contain a peritoneal sac and constitute a source of danger to the surgeon.

Sarcomas represent 24.7 per cent. of cord tumors. The clinical picture is similar to that of teratoma testis, and the tumor apparently arises from benign conditions. Fibromas of the cord arise in most instances from the cord near its junction with the epididymis, occurring about one-third as often as lipomas. Myxomas and leiomyomas are extremely rare in the cord. Dermoid cysts are of approximately the same frequency as fibroma. They are all simple in type.

In the epididymis, sarcoma is most common; ten authentic cases are reported: one case of lipoma, six of leiomyoma, and four of carcinoma, including a personal case. No dermoids, fibromas, or myxomas have been found.

Four cases of fibroma of the tunica albuginea are reported. No sarcomas are found. One instance of lipoma of the tunica vaginalis, thirteen cases of fibromas, two of rhabdomyoma, and twelve of sarcoma are on record. Epithelial tumors occur rarely, two cases of adenomatous tumors and one lymphangio-endothelioma having been found in this review.

The authors consider that benign tumors should be accorded the treatment of local excision, whereas malignant tumors, since they follow the course of testicular tumors and metastasize to the retroperitoneal lymph nodes along the aorta and vena cava demand radical treatment with resection of the regional lymphatics.

[Ed. Note.—The preponderance of malignant tumors in the spermatic cord, epidymis and testicular tunics (if we exclude the

54. Hinman, F., and Gibson, T. E.: Tumors of the Epididymis, Spermatic Cord and Testicular Tunics: A Review of the Literature and Report of Three New Cases, Arch. Surg. **8**:100-138 (Jan.) 1924.

fibromas) is worthy of emphasis, as in dealing with swellings in this region, we are too prone to consider the condition benign. This report confirms the need for a pathologic examination of every tumor removed from the inguinal canal and scrotum in order that the best end results for each patient may be obtained. Clinical differentiation of tumors is difficult and at times impossible, even in the hands of the most experienced, and again we see brought to light the constant need of cooperation between the surgeon and the laboratory in seeking for the truth.]

The work of Fuller, Squier, and others, in the development of operations on the seminal vesicles, is reviewed by Morrisey and Smith.[55] Cunningham's classification of inflammation of the vesicles into acute and chronic suppurative types is followed. If pain was the predominating symptom, with pus, epididymitis, and prostatic infection, seminal vesiculotomy consistently gave good results. The pain readily disappeared, the urethral discharge diminished, and the prostate returned to normal size. The group of rheumatic patients with suppurative seminal vesiculitis did not show such prompt response to vesiculotomy. Joint pains and vague muscle pains disappear much more slowly.

[ED. NOTE.—It is in the prostatic tissue and the seminal vesicles that the residuum of gonorrhea finds its stronghold. Consistent massage of the prostate and stripping of the vesicles carried out at repeated intervals will, in most cases, tend to relieve the condition. There are certain cases, however, such as Morrisey described, in which, owing to fibrosis, the vesicle is cut off from its proper drainage into the ejaculatory ducts. Distention of such vesicles will obviously be an active source of infection to joints and muscles; removal or drainage by surgical means is indicated. Similarly, old chronic cases in which the walls are thickened, having vesicular compartments which are shut off into small cystic areas, are difficult to cure without surgery. Seminal vesiculectomy is an extensive operation requiring the knowledge of anatomy that is necessary for perineal prostatectomy.]

### EXSTROPHY OF THE BLADDER

A case of complicated exstrophy of the bladder in an infant which died twenty-three days after birth is reported by von Geldern.[56] A large and fungating red tumor below the umbilicus, a marked spina bifida, absence of an anus, undescended testes, opening of the small intestine onto the central portion of the exstrophy, and a rudimentary penis were noted. The scrotum divided itself into two parts with an intervening

55. Morrisey, J., and Smith, F.: Surgery of the Seminal Vesicles: Indications, Technic, Results, Surg., Gynec. & Obst. **37**:480-489 (Oct.) 1923.

56. Von Geldern, C. E.: The Etiology of Exstrophy of the Bladder, Arch. Surg. **8**:61-99 (Jan.) 1924.

space continuous with the perineum. The duodenum was short, and its third portion failed to extend across the vertebral column. The large intestine was absent. The vasa deferentia joined and emptied into the ureters about 1.5 cm. above their termination. Several diverticula from the exstrophied bladder were noted.

In these complicated exstrophies of the bladder, there is evidence of a retained cloaca. A detailed analysis of similar cases is given. The theories of causation are classified as mechanical, pathologic, or due to arrested development. The mechanical theory comprehends a rupture or "burstungs" theory, explaining exstrophy as due to a rupture of retained fluid in the bladder. A short or absent umbilical cord may be a factor, as also may be an abnormal position of the vitellin duct.

According to the pathologic theory, exstrophy is due to an ulceration of the abdominal wall with subsequent necrosis and separation of the symphysis and underlying parts.

The theory of arrested development takes into consideration other malformations so often seen with exstrophy, such as cleft palate, spina bifida, umbilical hernia, and defective genitals.

Von Geldern believes that there is a strong similarity in complicated cases of exstrophy. The experiments of Morgan and Hertwig, who produced spina bifida by subjecting frogs' eggs to the action of 0.6 and 0.7 sodium chlorid solution, suggests that some chemical change in the uterine mucosa may be the inciting cause of exstrophy and allied malformations.

Simple exstrophy represents a differentiation of the embryonal structures from the normal, with cleavage of the cloacal membrane above the cloacal tubercle. This abnormal development depends upon the presence of some chemical substance in the uterine mucosa, and the degree of the anomaly, whether it be simple or complicated, is thought to depend upon the strength of the chemical agent or the resistance of the embryo.

[ED. NOTE.—The theory of arrested development has been most emphasized, especially among clinicians. The idea of maldevelopment due to biochemic forces is an excellent one. It brings to mind Loeb's experiments with the fertilization and development of amphibian eggs in vitro. The author's reasoning is highly technical. It is a suggestion which must be subjected to much study in comparative embryology and the biochemistry of tissue growth before it can have a definite practical significance.]

### ANESTHESIA IN UROLOGY

By the sacral and caudal methods a maximal anesthesia in the anoscrotal penile, sphincter ani and gluteal regions is obtained in about twenty-five minutes. Seventy-six patients were operated on under

sacral anesthesia by Lowsley.[57] The operations were fifty perineal prostatectomies, five seminal vesiculectomies, three Young punch operations, five urethrotomies, and so forth. All but six patients were satisfactorily anesthetized. Several patients had slight transient reactions. The postoperative course following sacral anesthesia is excellent, the shock of the entire body mechanism being considerably reduced by avoiding inhalation anesthesia.

[ED. NOTE.—The introduction of sacral anesthesia has proved a distinct advance in genito-urinary surgery. Crowell has reported a series of cases of perineal prostatectomy performed under caudal anesthesia, and the use of sacral anesthesia combined with lower abdominal field block has proved entirely satisfactory in the hands of Judd, Hunt. and others, in the performance of suprapubic prostatectomy. In certain clinics the procedure has been adopted as a routine. A point to be emphasized is the danger of penetrating the dural sac in inserting the needle in the sacral hiatus. The needle should not be advanced more than 4 or 5 cm. into the sacral canal, and aspiration with an empty syringe should be carried out to eliminate the possibility of the needle being in the dural sac or one of the sacral veins. With these precautions the method is relatively safe. In an average of fourteen dissections, Thompson found that the end of the dural sac lay 5.8 cm. from the sacral ligament, the longest sac being 7 cm., and the shortest 4 cm.]

### URINARY INFECTION

Hewitt[58] reviewed thirty-four cases of pyelitis complicating pregnancy and the puerperium. No cystoscopic examinations were made. The majority of the patients were between twenty and twenty-five years of age, and fifteen of the thirty-four were primigravidae. Only two patients had symptoms prior to the fifth month. Hewitt divides his cases into acute, subacute, and chronic, and discusses the symptoms and diagnosis of the condition. In none of the acute and subacute cases did pregnancy continue to full term, and in five of thirteen mild cases, pregnancy terminated before full term. In four cases, labor had to be induced. Such results were so unsatisfactory that alkalis were administered as advised by Kidd. Doses of 60 gr. soda bicarbonate and 20 gr. of potassium citrate were given at two-hour intervals for several days. In the fifteen cases in which this treatment was given, favorable results were obtained, there being only one premature labor, and in that case a living child was obtained. In three cases, there was a recurrence of

57. Lowsley, O. S.: Major Urological Surgery Under Sacral and Parasacral Anesthesia, Surg., Gynec. & Obst. **37**:688-692 (Nov.) 1923.

58. Hewitt, J.: Bacterial Infection of the Urinary Tract Complicating Pregnancy and the Puerperium, J. Obst. & Gynec. Brit. Emp. **30**:390-413, 1923.

pyelitis during the same pregnancy. Hewitt observes that recurrences are relatively common and that premature labor is more prone to occur during recurrence.

[Ed. Note.—The treatment of pyelitis of pregnancy as outlined by the author, by drugs only, appears to be quite unsatisfactory, and the number of premature and induced labors unusually high. The results of ureteral catheterization and lavage of the renal pelvis have been so satisfactory that this has almost become the standard method of treatment in this country.]

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# THE SURGICAL TREATMENT OF MITRAL STENOSIS: EXPERIMENTAL AND CLINICAL STUDIES *

ELLIOTT C. CUTLER, M.D.; SAMUEL A. LEVINE, M.D.

AND

CLAUDE S. BECK, M.D.

BOSTON

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* From the Laboratory of Surgical Research, Harvard Medical School, and the medical and surgical clinics of the Peter Bent Brigham Hospital.

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## I. INTRODUCTION

Surgery of the heart, even in its original and simplest form, that dealing with the treatment of traumatic wounds, is still in its infancy. Long before the function of the heart was known (1628),[1] the frightful mortality following wounds of this organ left the ancients[2] with the deeply rooted conviction that any injury to it would necessarily prove fatal. This impression existed up to quite modern times. When Paré[3] described the case of a man who received a wound of the heart in a duel and ran after his adversary 230 yards before succumbing to the injury, the learned societies of his times were greatly perturbed by this almost unbelievable observation. Still more recently (1883), Billroth remarked that no surgeon who wished to preserve the respect of his colleagues would ever attempt the suture of a wound of the heart, and in 1884 Riedinger[4] stated that the proposition to suture a wound of the heart although made in all seriousness, need hardly be mentioned.

It is unnecessary to dilate on the dangers accompanying surgical intervention in cardiac conditions. The heart is a vital organ, relatively inaccessible, and its state of constant activity creates a condition that raises the technical consideration of any operative intervention to the level of prime importance. It contains, moreover, the maximum capacity for hemorrhage, that most terrifying accompaniment of surgery. Such considerations doubtless prompted Billroth's statement. However, the extraordinary development of the surgical method of treatment since the introduction of asepsis has given our generation tools, methods, and an understanding that nullify comparison with past efforts. Even so, the application of surgical methods to the treatment of cardiac conditions remains practically restricted to the care of wounds of the heart, and in this field itself the available knowledge is only poorly disseminated.

This is unfortunate, since there may exist cardiac conditions for which surgery may be the only effectual treatment; and since the dangers are chiefly those of technic, they should be surmountable. With the hope of accomplishing this we have sought to expand the scope of surgery of the heart by the elaboration of a method to meet the proposal[5] that certain cases of mitral stenosis may be benefited by a surgical enlargement of the narrowed orifice. If such a procedure could be established, it might well follow that other mechanical abnormalities of the heart, both congenital and acquired, would also be amenable to surgical treatment.

## II. THE RATIONALE FOR OPERATIVE INTERVENTION, THE DIAGNOSIS OF MITRAL STENOSIS, AND THE SELECTION OF CASES FOR POSSIBLE SURGICAL TREATMENT

**Mitral stenosis is not only one of the most distressing forms of cardiac disease, but in its severe forms it resists all treatment by medicine.—*Brunton.***

One might reasonably question the wisdom of surgical intervention in cases of mitral stenosis on the ground that the symptoms are not due to the mechanical obstruction at the orifice but to the weakness of the heart muscle. Some years ago, physicians were likely to attach great importance to heart murmurs and to the functional condition of the valves. In more recent years, especially as a result of the teachings of Sir James Mackenzie,[6] all attention was directed to the heart muscle. In fact, one might be led to feel that whether the valves were normal or abnormal was practically of no significance. We think that the pendulum has swung over much too far in this controversy. There are many hearts which, at necropsy, show essentially normal musculature in the ventricles, but markedly deformed valves. It is hard to believe that the mechanical embarrassment to the circulation resulting from the contracted "fish-mouth" mitral orifice (Fig. 14), played no significant rôle in the production of the symptoms of cardiac failure. In fact, it is not unlikely that the heart muscle itself may suffer as a result of the mechanical obstruction, aside from the effects of the original condition that injured both the valves and the muscle. It seems logical to believe that the general and pulmonary circulation would be decidedly improved if a markedly constricted mitral orifice were enlarged.

No one who has seen any number of young adults suffering from mitral stenosis and has watched them go through several attacks of cardiac failure only to die finally after a lingering illness, can help being impressed with the inadequacy of medical treatment; and, when one finds at postmortem examination valves that are markedly contracted and that were mechanically impeding the normal circulation, at least one important reason for our failure in treatment becomes apparent. The question, therefore, arises as to whether the surgical approach to this condition is possible and whether or not improvement in the health of the sufferer will result if the stenosis is reduced.

It has been recognized since 1715 (Vieussens) that with mitral stenosis there results an increase in the resistance to the blood flow from auricle to ventricle, thereby limiting the filling and reducing the output. The factors concerned have been repeatedly studied. MacCallum[7] showed that by obstructing the flow at the mitral orifice with a clamp there results a fall in arterial pressure and a rise in the pres-

sures in the pulmonary circuit. Bernheim,[8] by placing and tightening a suture placed about the base of the mitral orifice, attempted to create a stenosis that could be later divided with an instrument, thus conducting an experiment similar to that which might occur if the operative method of treatment were applied to human cases. Allen and Graham[9] repeated similar investigations with pressure studies.

We have performed physiologic experiments and produced acute mitral stenosis by the same method, and have been able to record the auricular and ventricular pressure changes under conditions simulating those which probably occur in human cases. The stenosis created by

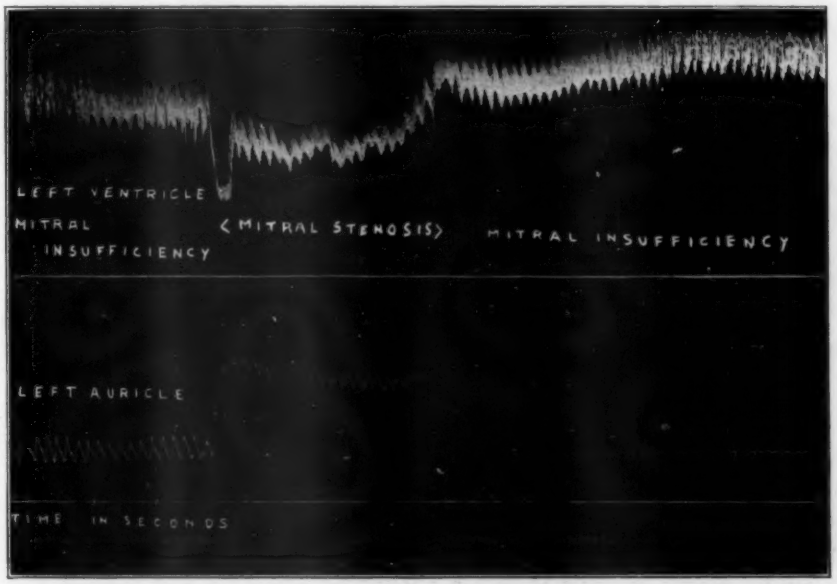


Fig. 1.—Pressures in left auricle and left ventricle of dog in which, after the establishment of mitral insufficiency, stenosis was produced by the use of a ligature at the base of the mitral ring. With the onset of stenosis there occurs a fall in the ventricular pressure and a rise in the auricular pressure; with the release of stenosis, the pressures return to their former level.

such experiments results in a rise in the left auricular pressure and a fall in the left ventricular pressure (Fig. 1). The rise in the left auricular pressure produces a stasis in the pulmonary circulation, the deleterious effects of which are commonly seen in human cases[10] and have been experimentally studied by Drinker, Peabody and Blumgart.[11]

In the past, it has not been of great practical importance whether the structural diagnosis in a patient suffering from cardiac failure was absolutely correct or not. The matter was somewhat of academic interest. Occasionally, the prognosis would be affected, but therapeutic

measures would always be directed in much the same way towards the phenomena of congestive failure, whether one valve or another were involved, and even when the valves were normal and the picture was altogether that of muscular insufficiency. But if surgical treatment is to have any success at all, the most accurate anatomic diagnosis must be made.

One item of great importance in the diagnosis of mitral stenosis is the past history of a rheumatic infection. This includes acute rheumatic fever, chorea, and acute tonsillitis with generalized pains in the limbs. The first two diseases are by far the most important.[12] In more than 50 per cent. of a large number of cases of mitral stenosis, one or the other of these two diseases will be found to be included in the past history. Of course, there will be large numbers in which this history is lacking. This is to be explained in a great measure by two factors: First, mild cases of acute rheumatic fever often go unnoticed, are called growing pains or the grippe by the mother, and even by the physician, pass without any one being aware that the affection was a smouldering rheumatic fever until years later, when the heart involvement becomes evident; secondly, mild cases of chorea are often overlooked and regarded by both parents and physicians as evidence of a nervous or fidgety child. Indications that both these mistakes have been repeatedly made has been observed in patients in the medical wards of the Peter Bent Brigham Hospital. It has frequently been our experience in both these two latent types of rheumatic affections to find slight, intermittent rises in temperature to 99.4 F. or 100 F. continuing over months or years, with the child even attending school and complaining little or not at all. In such cases, there may be an occasional pain in a limb or a slight twitch in the face or hands. It is likely that there is no other cause of mitral stenosis except a rheumatic affection, but we are not always able to discover this fact through questioning concerning the past history of patients. Sometimes contributary facts, such as a family history of rheumatic disease or valvular disease or a past history of repeated unexplained epistaxis in the individual, supply additional proof of the likelihood that a rheumatic infection is the underlying cause of the heart picture.

The symptoms of heart failure in general apply to all types of heart disease, and will be of no great assistance in distinguishing mitral stenosis from other affections. We are not greatly concerned in this paper with the question as to whether or not the patient has heart disease; to do this one would need to go into the general symptomatology of heart failure. After the diagnosis of heart disease has been made, the question arises as to whether or not we are dealing with a case of mitral stenosis. The high color of the lips and cheeks and the small

volume of the pulse are generally accepted findings, but they are not in the least pathognomonic. A direct examination of the heart itself is of more value. Enlargement of the heart by percussion is common in all types of heart disease. The presence during diastole of a thrill that can be felt at the apex is of great importance, and this is frequently found when the constriction of the valve is considerable, and the circulation is still in fair condition. The thrill may, however, be absent in some cases in which there is a high degree of mitral stenosis. On auscultation, the accentuation of the first heart sound as it is heard at the apex is extremely valuable. This occurs early in the development of mitral stenosis and is apt to persist until the heart muscle has become considerably weakened. It must be borne in mind that such accentuation of the first heart sound also occurs in some nervous patients in whom there is no heart disease, as well as in many cases of hyperthyroidism.

The critical point in the diagnosis is represented by the detection of a murmur in diastole. In fact, in many cases the diagnosis cannot be made without auscultation, and in some patients, any kind of heart disease might not otherwise be suspected. When there is only a slight degree of stenosis the murmur is likely to be short, generally presystolic in time, crescendo in quality, ending with the snapping, first heart sound. At times, the murmur is rumbling in quality and is heard in middiastole. These murmurs are best heard at the apex of the heart; in most cases, a systolic murmur may be heard as well, although this is not always the case. As the stenosis becomes more marked, if the musculature still remains fairly competent, the murmur lengthens out so that finally it can fill the entire diastolic period. It is assumed all this time that the rhythm of the heart continues essentially regular, although there may be interruptions by extrasystoles arising in one or another part of the heart.

Somewhere along the course of its development auricular fibrillation is likely to set in. Although it generally occurs from fifteen to twenty or more years after the occurrence of the original rheumatic infection, there is great uncertainty concerning it. Sometimes, the auricles start fibrillating a few years after the first infection, with only a moderate degree of stenosis; in other cases, the interval between the original rheumatic attack and the development of auricular fibrillation covers many years. The type of diastolic murmur that persists with the change of rhythm from the regular to the absolutely irregular will depend somewhat on the degree of stenosis present when the change occurred. If the entire diastole has been filled with the murmur when the heart was regular, a long diastolic murmur will be heard when the heart is fibrillating, except that there will be no presystolic accentuation. In order that the long diastolic murmur may be heard the heart rate must be slowed; otherwise, the pause between heart beats will be too short.

In fact, one may even be led to think that the presystolic element has persisted with auricular fibrillation when that is not the case. On the other hand, if there has previously been a short, late, diastolic murmur when the heart was regular, most of it may disappear when auricular fibrillation sets in. In general, it may be said that the longer the diastolic murmur, other things being equal, the greater the degree of stenosis. There are occasional exceptions to this, especially in advanced cases in which very marked stenosis may be present at the same time that the diastolic murmur is very short or even inaudible.

There are other means at our disposal that help considerably in the making of the diagnosis. A roentgenographic study of the heart is important,[13] and especially so is the finding of a prominent, left auricle in the ordinary 7 foot roentgen-ray plate of the heart (Fig. 2). Often, the bulging of the left auricle is more evident posteriorly than to the left and can be best made out fluoroscopically. The enlargement of the heart shadow to the right of the midsternal line is a frequent finding in mitral stenosis; but it is not of great assistance in diagnosis, because such enlargement may occur in cases of chronic myocarditis without disease of the valve, and in adherent pericardium. Finally, electrocardiograms throw considerable light on the diagnosis. It is common to find peculiar prominent auricular or P waves that are notched and flat-topped. In addition, there is apt to be evidence of right ventricular preponderance, which is indirect evidence that there is a lesion of the mitral valve (Fig. 3). This last criterion is not at all crucial in its application and must be carefully interpreted with the other findings.

When all the factors that may have any bearing on the diagnosis are properly appraised, the correct conclusion can be reached with a high degree of accuracy. Of greatest importance is the diastolic murmur, with or without a diastolic thrill at the apex. Second to this is the presence of a prominent left auricle as indicated by the roentgen-ray examination and the changes in the electrocardiograms.

The difficulties of the future will be encountered in the attempt to distinguish the cases of pure mitral stenosis from those in which there is in addition tricuspid and aortic valvular disease. The diagnosis of adherent pericardium is very difficult, but it will obviously be detected by the surgeon when the chest wall is opened. In this way, the proper operation such as a cardiolysis can be performed, whatever the preoperative diagnosis may have been.

In the selection of suitable cases for surgical intervention, there are several factors to be borne in mind. It would be preferable to choose those cases in which the patients are beyond the age in which repeated rheumatic infections are likely to occur. It is well known that children are prone to recurrent attacks of rheumatic fever, even when the customary precautions are taken. This means that the infection is still

Fig. 2.—Seven foot heart plate from a typical case of mitral stenosis showing the prominence of the left auricle. It is important to distinguish this prominence from that due to the pulmonary artery which lies at a higher level.

Fig. 3.—Electrocardiograms of a case of mitral stenosis; three customary leads. Timer below indicates fifths of a second. Standardization (3 millivolts = 3 cm.), is shown at the beginning of each lead. The prominent, notched, and flat-topped auricular complexes or "P" waves in leads 1 and 2 may be observed. There is a tendency to right ventricular proponderance.

latent or, at least, that it might undo any improvement that would result from operation. Such recurrences are less likely to occur after the age of 20. It is also wise to select those cases in which the mechanical construction of the valve is quite marked. The physical examination of the patient and the detailed study of all phases of the problem discussed above will help toward arriving at the proper estimate of this factor. Finally, one should try to select those patients in whom the musculature might be expected to be in fair condition. Here again, proper appraisal can only be made as a result of experience in which all methods of study are utilized. In this connection, electrocardiograms that indicate a healthy ventricular musculature will be of value.

## III. HISTORICAL DATA

It is always of interest and sometimes of great value to examine the historical background of any new undertaking. In this instance, it is not only valuable but necessary, since it is only by a review of the many sources from which not only we, but also our predecessors in this field, have drawn inspiration, knowledge, and mental equipment, that one can appreciate the opportunity that made possible these investigations. The opportunity arose as the result of a prolonged and laborious but steady development in surgical technic, the adaptation of physiologic methods, and the final recognition of the desirability of the procedure.

A. *The Beginnings of Cardiac Surgery: the Suture of Wounds of the Heart.*—The surgical advance in the care of wounds of the heart is a modern development. It has already been stated that the ancients accepted as final the fact that any wound of the heart was necessarily fatal; this, too, long before the function of the circulatory apparatus was appreciated. It was not until Fischer's splendid monograph [14] on wounds of the heart in 1867 that any general recognition was given to the opinion that cases of wounds of the heart were recoverable. Fischer stated that about 10 per cent. of all heart wounds healed safely. His opinion was derived from the postmortem finding in both animals and man, of healed wounds of the heart often containing arrow heads and other missiles. Unquestionably, the heart had been previously exposed in a search for missiles of war as exampled by Larrey's report in 1810 of a pericardiotomy for a wound of the pericardium and lung.[15] Such attempts, however, were sporadic and had little influence. From the time of Fischer's publication until 1884, when Rose [16] took a firm stand in favor of surgical intervention in cases of wounds of the heart, there was no progress in this field. Billroth, during this period, dominated European surgery, and his opposition to the surgery of the heart and pericardium has already been mentioned. Rose demonstrated the importance of "heart-tamponade" upon which he felt that the future of surgery of the heart depended. His ideas regarding the deleterious effects of an increased pressure in the pericardium had been previously described by Morgagni [17] in 1761 and by Franck [18] in 1877.

Experimental investigations into the possibilities of suturing wounds of the heart began about this time. Bloch,[19] in 1882, was the first to demonstrate successfully the feasibility of this procedure in animals, and he boldly proposed the same procedure in man. There were no further successful demonstrations in this field until those of Del Vecchio [20] in 1894, Salomoni [21] in 1896, and Bode [22] in 1897.

Meanwhile, the suture of wounds of the heart had been applied in human cases. Cappelen,[23] in 1896, reported an unsuccessful attempt, the patient dying two and one-half days after the operation from sepsis and pericardial effusion. The same year Durante, in a discussion of Salomoni's paper before the Eleventh Congress of the Italian Surgical Society, referred to a similar unsuccessful attempt by Farina.[24] The next year (1897), Rehn [25] reported the first successful suture of the human heart. Following this date, the number of successful attempts rapidly increased. Ten years later Rehn [26] was able to collect 124 cases in which operations had been performed for cardiac wounds, with 40 per cent. recoveries resulting. Tuffier,[27] in his address on the surgery of the heart before the Fifth Congress of the International Society of Surgery in 1920, assembled 305 cases from all sources, with 50.4 per cent. recoveries. Thus in fifty years, because of the application of surgery to the treatment of wounds of the heart, the patient's chance of living was raised from 10 per cent. to 50 per cent.

During all this period, animal investigations into methods designed to perfect surgical operations on the heart were carried on. From ancient times, the heart had been thought to contain vital centers, injuries to which caused instant death. The physiologists have made clear the importance of the region of sino-auricular node and the His-Tawara bundle. Kronecker [28] and Schmey [29] demonstrated in rabbits and dogs the existence of a small area in the interventricular septum, the puncture of which caused immediate cessation of the action of the heart. In an attempt to study further the effect of injury of the heart, Elsberg [30] conducted an admirable series of experiments in which he excised the apical third of the heart in dogs and rabbits and demonstrated the adaptability of the heart to extensive surgical procedures.

From the beginning, the control of hemorrhage occupied the minds of surgeons interested in this field. Among others, Rehn and Sauerbruch [31] independently conducted animal experiments designed to demonstrate how long the circulation could be clamped off, in order that the process of suture could be performed in a bloodless field. They showed that, in the dog, the entire pedicle of the heart can be clamped for about one minute with recovery. They also found that, if only the cavae were clamped, little bleeding occurred from the opened heart, but that the interval of time could be lengthened. As a result, they have advised digital compression of the cavae during the process of the suture of actively bleeding cardiac wounds. Laewen and Sievers [32] conducted similar experiments and obtained similar results.

B. *The Beginnings of Pulmonary Surgery.*—Any procedure that deals with an intrathoracic organ will depend to a certain extent on the development of surgery in relation to other intrathoracic viscera. Thus,

our appreciation of the best method of exposing the heart depends not only on the advances inherent in the development of cardiac suture for traumatic wounds, but also on the knowledge acquired in the attempt to alleviate certain pulmonary conditions by surgical treatment. Pulmonary surgery in its simplest form, the drainage of an empyema cavity, is as old as medical records. Hippocrates [33] advised it. It was, however, considered a dangerous procedure, and was but little practised until modern times. There do exist case reports of instances of hernia of the lung following trauma, with amputation of the protruding viscera with the cautery, healing of the wound, and recovery. Unquestionably, collapse of the lung when the chest was opened was the prime deterrent to further operative procedures, and this field of surgery had to await the development of a proper mechanical respiratory apparatus for administering anesthesia before any real progress could be made. The suggestion of Forlanini [34] in 1882 that pulmonary tuberculosis could be helped by the collapse of the lung on the affected side through the intrapleural injection of nitrogen gas, and J. B. Murphy's investigation [35] (1898) along the same line together with his enthusiastic reports in regard to the possibilities of surgery in other pulmonary conditions were a great stimulus to those interested in the surgery of the lungs. The Schede [36] and Estlander [37] (1879) external collapsing operations had already been proposed, and in 1893 and 1894, Fowler [38] and Delorme [39] had independently described their operations for decortication of the lung.

C. *The Development of Apparatus for Mechanical Respiration.*—These developments in pulmonary surgery, however, were limited by the fear of the danger that accompanied collapse of the lung. Thus, the procedures developed were either extrathoracic (Schede, Estlander), or adapted to intrathoracic conditions in which adhesions had already formed, and in which collapse therefore would not occur (Delorme, Fowler, Murphy, etc.). The first attempts to counteract pulmonary collapse during operative procedures were made by Quenu [40] and Tuffier [41] (1896). The former independently developed a helmet that could be fitted snugly over the patient's head and in which a positive pressure was created. The latter introduced a tube into the larynx and trachea and blew air in with a bellows, thus utilizing the method of insufflation later perfected by Meltzer and Auer.[42] Matas [43] in 1899 introduced a similar method in this country, and Parham [44] in 1898 removed a tumor of the chest wall using such an apparatus, thus performing the first intrathoracic operation on record in this country. F. T. Murphy [45] of Boston in 1905 proposed a positive pressure box similar in purpose to Quenu's method.

Another step, with the same purpose in mind, was the introduction by Sauerbruch,[46] in 1902, of a chamber in which the patient, operator, and assistants placed themselves, and in which a negative pressure was maintained. Only the patient's head protruded from this box, thus permitting a differential negative pressure, sufficient to expand the lungs. This apparatus, however, was both expensive and unsatisfactory in that it separated operator and anesthetist. In 1909, Meltzer and Auer introduced their simple intratracheal insufflation apparatus by which the anesthetic vapor is blown in under sufficient pressure to keep the lung expanded and with regular interruptions of the pressure to provide for proper ventilation of the lung. This method superseded all others and at once opened up great possibilities in thoracic surgery.

D. *The Brauer Operation.*—In 1902, Brauer, professor of medicine in Marburg, published the proposal for the operation of cardiolysis [47] in cases of adherent pericardium. At the same time, successful results in actual cases were enumerated. This undertaking was a step of great importance and is but one of the many brilliant examples of the method by which advances are achieved by workers who confine themselves to a field of medicine rather than, as is commonly the course, to a method of therapy. Although at that date surgery was not so highly technical as it is now and had not entirely broken away from its traditional place as the handmaiden of medicine, it certainly was unusual for a professor of medicine to formulate proposals for surgical operations. This step, moreover, was of importance as a further stimulus to the hope that surgery was not absolutely contraindicated in cardiac conditions. The satisfactory statistics now available in cases in which Brauer's procedure has been carried out do great honor to the originator of the idea; the experience, both technical and objective, has proved to be of considerable value in the more recent developments of surgical methods in cardiac conditions.

E. *Sir Lauder Brunton's Proposal.*—It is unnecessary to emphasize the importance of Brunton's proposal. When, in February, 1902,[48] his note appeared with the suggestion that, "the good results that have been obtained by surgical treatment of wounds in the heart emboldens one to hope that before very long similar good results may be obtained in cases of mitral stenosis," he courted criticisms for his temerity. Indeed, criticisms he received in full, the Lancet [49] even attacking his proposal in a sarcastic editorial. It seems to us that the chief objection proposed in the editorial mentioned was on technical grounds. From our perspective, it is difficult to judge with an appreciation of the feeling of that day; but as a matter of principle, if technical difficulties alone beset a problem, that is no indication for hesitation. The fact that most technical difficulties are surmountable with practice has been shown in

this case. That the technical difficulties in Brunton's day seemed enormous has been related to us by his surgical colleague, Sir D'Arcy Power, and further light is thrown on this by a communication from Sir James McKenzie, who states that he considered this proposal carefully but felt that he "would rather leave the troubles and the dangers for others." A storm of letters and short communications both condemning (Shaw [50] and Fisher [51]) and supporting his contention appeared. Tollemer of Paris [52] supported the idea, but set forth serious doubts as to whether the small knives that Brunton proposed using would be efficacious. Interestingly enough, our experience, to be revealed later, proved the correctness of these doubts. In addition, however, the criticisms, letters, and communications that appeared showed that the proposal was not new. Arbuthnot Lane,[53] some years previously, related his attempts to find a satisfactory case for the procedure proposed later by Brunton; and Samways [54] pointed out that, in an article of his published four years previously, he had made an identical proposal, viz.: "I anticipated that with the progress of cardiac surgery some of the severest cases of mitral stenosis will be relieved by slightly notching the mitral orifice and trusting to the auricle to continue its defense." Indeed, the suggestion is one that must have come to many a medical man, once Harvey had established and made known the function and action of the circulation. No frequent observer at necropsy could fail to observe the apparent ease with which a stenosed mitral orifice might be divided.

It is of great interest, therefore, to realize the status of surgery at the time Brunton made his proposal. The advances in surgery of the heart itself, especially those related to the proper handling of traumatic wounds, the advances in intrathoracic surgery, and the beginnings of the development of a successful mechanical respiratory apparatus for anesthetizing patients with the thorax wide open, are the best evidence that this suggestion came at a fortunate time. Certainly, surgeons were becoming more familiar with thoracic conditions, and the satisfactory method of administering anesthesia, shortly to be introduced by Meltzer and Auer, resulted in a tremendous impetus to experimental intrathoracic work. As Brunton said, the operation he proposed could not be undertaken without attempts first being made with animals. For this experimental work, though curiously not for the later development of the procedure in man, a mechanical respiratory apparatus was necessary for producing anesthesia and for protection against collapse of the lungs. Moreover, the year following the publication of Brunton's paper, Brauer's description of the operation for cardiolysis gave further impetus to the work in this very field. Almost immediately after the proposal of Brunton, surgeons began animal investigation into the possibilities of developing an operative method applicable to man.

F. *Physiologic Methods for Producing Lesions in the Valves of the Heart.*—Proper acknowledgment should be made to those investigators, chiefly physiologists, who had, long previous to Brunton, elaborated methods for the creation of valvular lesions, because surgical investigators, in putting Brunton's suggestion to actual experimentation, adopted the same methods. It is of interest to note that the first experiments recorded, in which valvular defects were created, were carried out by an ophthalmologist. Becker,[55] in 1872, in an exhaustive treatise on the pulsation of retinal vessels in aortic insufficiency, describes the manner in which, by the insertion of a glass rod down the left carotid artery of dogs, he was able to destroy one or more of the aortic cusps and to produce an insufficiency with retinal manifestations. This investigator, in turn, refers to Quincke before him as having laid bare the heart and produced an insufficiency by tacking back a cusp of the aortic valve resulting in the production of a murmur. In 1875, Klebs [56] conducted experiments similar to those of Becker, using a tiny knife on a long rod which he called a "valvulotom" and which he introduced down both the jugular veins and the carotid arteries at the base of the neck. He was able to create defective valves and keep the animals alive. Cohnheim [57] in 1877 described the use of sounds made of whalebone similarly inserted down the carotid arteries in the neck and used to tear out cusps of the aortic valve; he was interested primarily in a study of the arterial pulse following these lesions. The next year, and again in 1889, Ottomar Rosenbach [58] conducted experiments, using the Klebs valvulotom and sounds in order to study the pulse and the heart action following the creation of an insufficiency in different valves. He attacked the aortic valve by way of the carotid artery, and the tricuspid by way of the jugular vein. In 1888, Timofejew [59] produced similar defects in the aortic valve by the use of needles and sounds inserted down the aorta in an attempt to correlate the size of the defect with the type of murmur produced.

Similar experiments using analogous instruments continued to be used by physiologists and internists in a study of circulatory changes with defective valves up to the present time (Kornfeld,[60] Hasenfeld and Romberg,[61] MacCallum [62] and McClure,[7] and Wiggers and DuBois [63]). MacCallum, in 1906, besides demonstrating interesting pressure changes with the use of such methods, presented the suggestion of the value of these experimental insufficiences in teaching medical students. In some of his experiments, instruments not dissimilar to the Klebs "valvulotom" were inserted into the heart through the auricles or ventricles, the method soon used by surgical investigators in their study to find a suitable operative method that might be adapted to the operative relief of mitral stenosis.

G. *Experimental Surgery of the Valves of the Heart.*—We have seen that intrathoracic surgery, at the time Brunton proposed the possibility of surgical intervention in cases of mitral stenosis, was making great strides. Indeed, it soon became a favorite field for surgical investigation. It was only natural, therefore, that the possibilities of cardiac surgery should share in the attempt more commonly aimed at advancing our knowledge and technic regarding pulmonary surgery. Moreover, as we have pointed out, the physiologists had already provided certain methods and instruments for creating valve lesions; there had already been considerable experimental investigation into the ability of the heart to withstand trauma, into its so-called vital areas, the course and similarity of the healing of wounds of the heart to wounds elsewhere, into the technic of the proper handling of the pulsating organ, and into the effect on its action of total and partial occlusion of the great vessels arising from it and entering it at its base. It seems, therefore, almost assured that, with or without Brunton's suggestion, some surgical investigators would have investigated their ability to create valvular defects with satisfactory implements and methods, with a view to operating on stenotic valves in man. The contributions of Haecker [64] in Germany and of Cushing and Branch [65] (in collaboration with Henry and Hauer) in this country, which appeared almost simultaneously in 1907, were the first reports of recovery experiments in which the heart valves were cut, a thoracic approach having been made. Both groups of investigators approached the subject not primarily to develop a technic for the surgical attempt on human cases of mitral stenosis, but out of a desire to study the remote effect of valvular insufficiency. They were, however, fully aware of Brunton's suggestion, and unquestionably looked forward to the future development of cardiac surgery. Later observers acknowledge their debt to MacCallum for the methods they used, thus giving direct evidence of the bearing of the early experiments of physiologists on the subsequent perfection of operative procedures. In their paper they not only describe the creation of insufficient valves by the use of small hooks, but they explain the method by which a temporary mitral stenosis may be created by tightening and tying a suture laid about the mitral ring in the subcoronary region. The contribution of Cushing and Branch is of particular importance because it was the first real proof that the operative creation of valvular defects could be carried out with a high degree of certainty, that each attempt would be successful and with a sufficiently low mortality (they reported eleven recoveries in twenty-five attempts) to hope that, with improvements in technic, the risk could be made almost negligible. Von Haecker's observations are far less impressive as only one animal in thirteen experiments survived the ordeal. However, many of his experiments were conducted with the entire circulation shut off by means of a clamp

at the base of the heart, after which the ventricle was opened and the valve cut with scissors. As a pupil of Sauerbruch, he doubtless used this method after having perhaps taken part in the earlier investigations of his chief into the question of the best control of hemorrhage when operations on wounds of the heart were being performed. Having performed similar experiments ourselves, we are not surprised at his meager success.

Bernheim,[8] in 1909, published a series of experiments in which it had been the purpose to create mitral stenosis by the use of a ligature

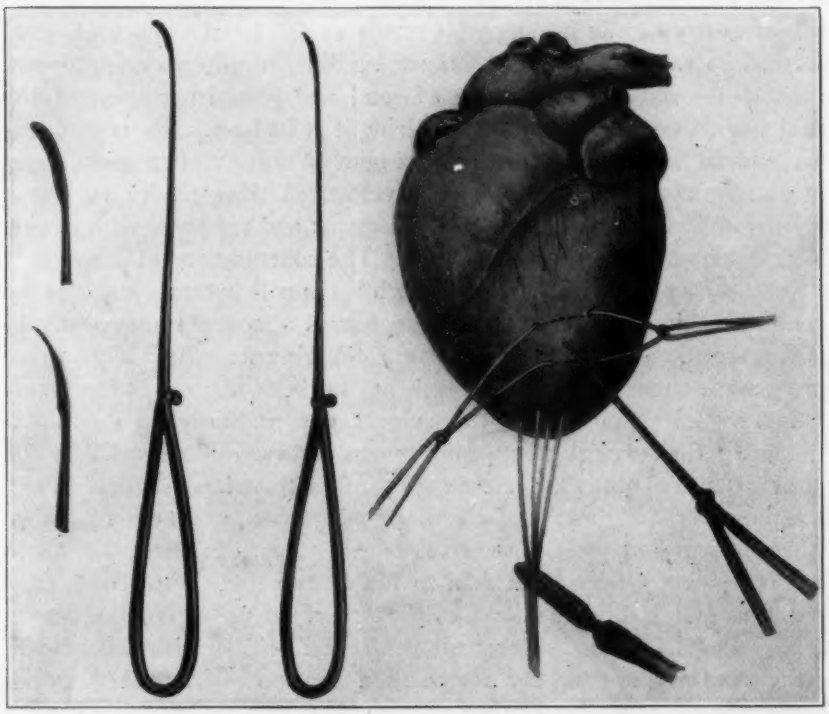

Fig. 4.—Reproductions from Schepelmann showing the knives and his procedure.

about the base of the mitral valve and then later to cut this and bring about a return to the normal condition, thus simulating what he hoped could be accomplished in the release of the stenosis in human cases. In only one experiment, did he succeed in accomplishing his purpose; the methods described, both for placing the suture and releasing the stenosis, are in principle similar to those used by Cushing and Branch. These attempts are the first surgical investigations undertaken with the primary intention and purpose to experiment with an operative procedure in

animals that might later be applied to man. It would be of interest to know why Bernheim did not continue his investigations, because he brings no important criticisms to bear either on the idea or on the execution of his attempts.

In 1912, Schepelmann [66] conducted various experiments designed to test the methods previously suggested for creating lesions of the heart valves. He repeated the procedure of Haecker, clamping off the heart at

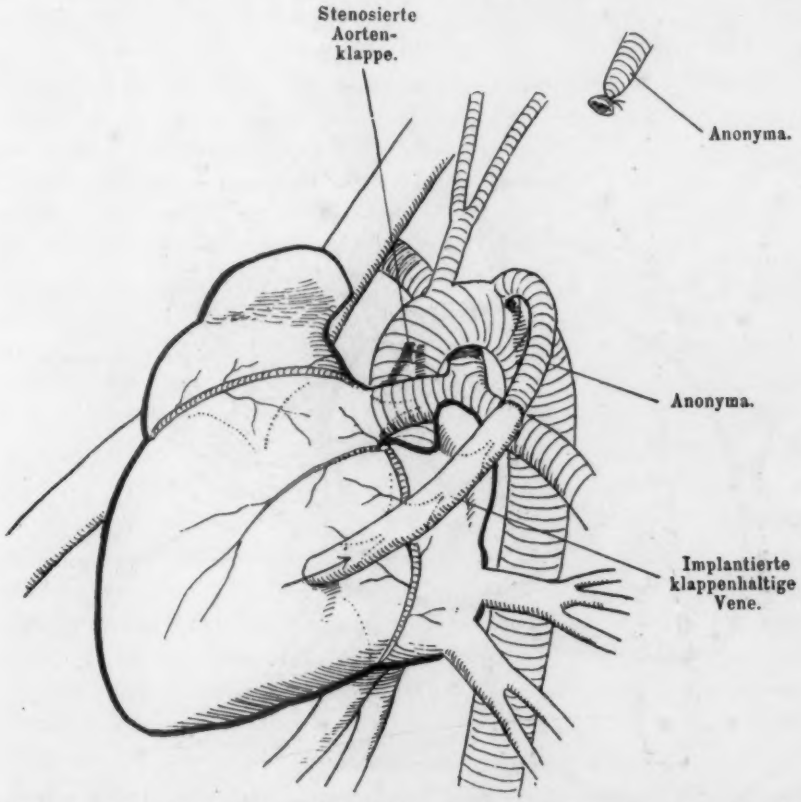


Fig. 5.—Reproductions from Jeger demonstrating his proposal that in cases of aortic stenosis the blood stream be shunted around the stenosis by the insertion of a segment of vein implanted in the left ventricle and connecting with the innominate artery.

the base, opening the ventricle and cutting the valves; he also inserted small knives (Fig. 4), on long shafts through the ventricular walls, and slit the valve cusps; he used a silver wire ligature beneath the mitral ring in his attempts to produce stenosis. No new suggestions were made as to improvements in methods previously used, although his long-handled small knives, which were of the general plan of the instruments

we first used, were better suited to their task than the hook knife used by MacCallum, Cushing and Branch, and Bernheim. No actual results are quoted, but he does not recommend such procedures in man because of the probability of disease of the heart muscle, although he states that they are feasible in animals.

The monograph of Jeger,[67] published in 1913, "Die Chirurgie der Blutegefasse und des Herzens," contains thirty-three pages devoted to experimental surgery of the heart. It is chiefly a historical review of

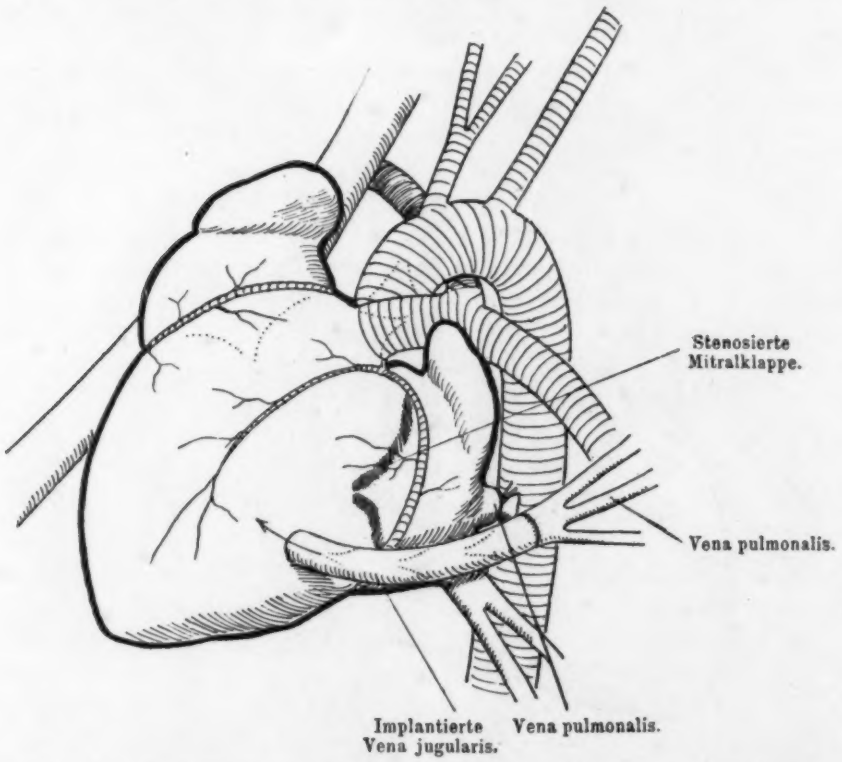


Fig. 6—Reproduction from Jeger demonstrating his proposal that in cases of mitral stenosis the blood stream be shunted around the stenosis by the insertion of a segment of vein connecting the pulmonary veins and the left ventricle. In both Figures 5 and 6 the segment of vein is so sutured that its valves open only in the desired direction.

the development of surgery of the heart, containing references to the experiments cited above. However, two interesting diagrams that embody a new principle are presented. He proposed that in aortic stenosis a new channel for the blood stream should be constructed by implanting a segment of jugular vein into the left ventricle and suturing its distal end to one of the first branches from the aorta

(Fig. 5). This procedure was carried out once in a dog, using the arteria anonyma, and the animal survived four days. At necropsy the vessel anastomosis was secure, but there was a fresh thrombus. There were also fresh, small clots in the left ventricle and in the mouth of the vein protruding therein. A similar procedure is suggested for mitral stenosis, the transplanted vein being anastomosed with the pulmonary vein and entering the ventricle in a similar manner (Fig. 6). Unfortunately, further experiments were not cited.

The many contributions of Carrel and Tuffier [68] contain much information of great value to workers in this field. They have well emphasized the necessity for technical precision and for perfect asepsis in intrathoracic surgery, and their results prove that, with such technical precautions, this field is as safe for surgery as any other. They review methods previously used and introduce a new method. This might be called an extracardiac method for producing valvular insufficiency. They first sutured a section of preserved vena cava over the region of the pulmonic orifice, tacking it down securely except at the inferior border; then, at this point, they slipped beneath the patch a pair of fine scissors, divided the tissue supporting the valves completely, held down the patch with forceps, extracted the scissors, and finally tacked down the inferior border of the patch. This resulted in a bulging outwards of the ring at the base of the valves and a resultant insufficiency of the cusps. They also attempted to cut and sew together the mitral cusps, with the circulation closed after the method of Haecker. In a discussion following the presentation of some of their work, when he was asked the question as to whether such operations could be done in human cases in which there was diseased muscle, Carrel replied that the question might be answered by experience, and that there were no methods for producing an experimental stenosis. However, they did think that a procedure that necessitated clamping off the entire circulation should not be used in cases in which the muscle was already damaged.

In 1922, Allen and Graham [69] presented their first communication, with the introduction of a new method. They stated that "the primary object of our experimental work was to develop a practical surgical procedure for the relief of mitral stenosis." They elaborated the advantages that would be present if the division of the valves could be done under direct vision, but they discarded the method of cutting the valves after opening the ventricle with the circulation cut off (Haecker, Carrel, and Tuffier). It seemed to them that the short period of time (not more than three or four minutes) available by this method would be insufficient for any satisfactory procedure. They constructed, therefore, a small endoscope carrying an electric light resembling in principle the

female cystoscope (Fig. 7). This was equipped with a small curved knife the handle of which protruded at the proximal end, and the distal end of the tube itself was given a semi-circular glass tip with a groove into which the knife fitted. The rounded glass end made it possible to place it against structures within the heart chambers which could be seen when close contact was thus etablished. They first inserted this instrument through the wall of the left ventricle, but because of a 50

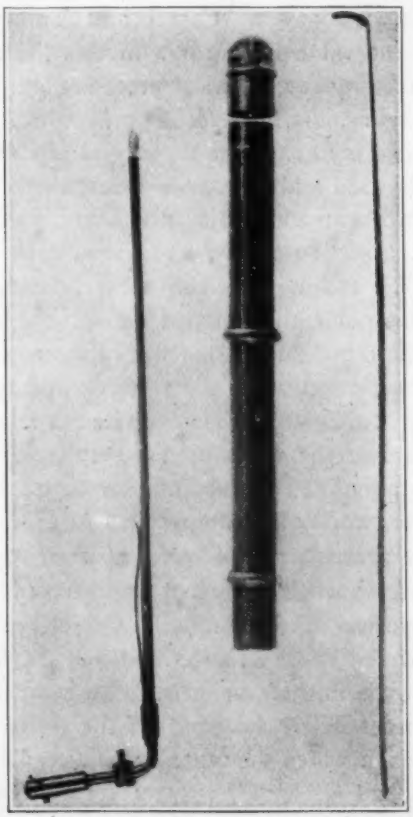

Fig. 7.—Reproduction from Allen and Graham showing their cardioscope.

per cent. mortality they tried the insertion of their cardioscope through the left auricular appendix. This route had been used by MacCallum, and the trauma produced here had no undesirable influence on the action of the heart. With this method, they were able to divide the normal valves under vision without interruption to the flow of the blood. They reported a low mortality at operation (one death in twenty-two operations), but there were many deaths subsequent to operation from sepsis. These investigators have also repeated the physiologic experi-

ments of recording pressure changes in the chambers of the heart, with and without a temporary string stenosis and with insufficiency.[9] They point out the beneficial pressure changes that accompany the reduction of a stenosis to an insufficiency.

An intimate study of any single field of endeavor affords an excellent picture of the changes taking place in the entire field. Thus, this review depicts the extraordinary advance made in surgery as a whole. It particularly emphasizes the interrelation of medical sciences in the obvious example portrayed here of the debt surgeons in this small field owe to the physiologists and internists; it is an interesting example of how various developments, when they reach a certain point, create an opportunity that is utilized by some one. The perfection of surgical technic, the increasing proficiency and accumulated knowledge of surgeons both in the treatment of wounds of the heart and in the field of pulmonary surgery, and the development of a reliable mechanical respiratory apparatus for administering the anesthetic were the chief elements in the development of a surgical method for the treatment of mitral stenosis. We now know that Brunton's proposal had been similarly suggested a little earlier, and the circumstances are such as to make one almost certain that it had been put forward many times and many years before his day. The criticisms of Brunton's proposal, however, were based not only on a prior argument as to the desirability of the procedure, but also in large part on the technical difficulties which in his day seemed insurmountable. These latter criticisms were mistaken in principle, because technical difficulties are rarely insurmountable with practice, ingenuity and the passage of time. The contributions of Haecker, Cushing and Branch, Schepelmann, and Allen and Graham disprove this latter contention. We find ourselves, therefore equipped with satisfactory methods for the creation of valvular defects in animals and with enough operative knowledge concerning wounds of the heart in man to make it seem feasible to practice these experimental methods in cases of mitral stenosis in man.

## IV. EXPERIMENTAL DATA: EARLY EXPERIMENTS

**But no one would be justified in attempting such a dangerous operation as dividing a mitral stenosis on a fellow creature without first testing its practicability and perfecting its technic by previous trials on animals.—*Brunton.***

It is difficult to analyze at this time the stimuli that led us to begin this investigation in 1920. Certainly, we were increasingly impressed with the inadequacy of medical treatment in many cases of mitral stenosis. The suggestion of Sir Lauder Brunton had been brought to our attention. The preceding historical review depicts how obvious was the opportunity. The proposal and useful methods for its study were at hand, and the technical improvements in surgery had advanced to the stage in which difficulties from this aspect alone no longer assumed the dominant rôle. The cranial chamber having been successfully brought into the province of safe surgery, chiefly through the technical methods of Professor Cushing and his pupils, it appeared that the heart alone remained as a field for the almost unchallenged labors of the internists. That this state of affairs was not to continue long was evidenced by the developments already stated.

Fortunately, one of us had already conducted experimental studies involving exposure of the heart of the cat.[70] In these experiments, levers had been sutured against the walls of the heart, and cannulas had been inserted into the various chambers without much disturbance of function, so that we began with no particular trepidation concerning the inability of the heart to withstand trauma and manipulation.

From the beginning, we set out to develop a surgical method for the treatment of mitral stenosis. It seemed to us, as has already been discussed in Section II, that the medical treatment of chronic cardiac valvular disease, and especially that of mitral stenosis, was inefficient. It was hoped and expected that the technical difficulties could be surmounted. This could only be accomplished by means of animal experimentation.[71]

A. *Operations with the Circulation Closed (Visual Method).*—We began with the proposition that division of the mitral orifice under direct vision was the most desirable method, and in spite of the failure of Sauerbruch, Haecker, Carrel and others to accomplish much with this method, which necessitated clamping off the general circulation, we began our work by repeating such investigations. We felt that, even if our attempts were unsuccessful, we would gain much by the familiarity acquired in handling the heart and through a further study of its resistance to trauma.

Cats were used in the first investigations. Ether was employed in all experiments, administered through an intratracheal tube. The thorax

was shaved and opened by a median incision that split the sternum, and avoided the mammary vessels. Retraction of this opening gave ample exposure in the cat. The pericardium was easily opened, and the heart delivered. For closing the circulation we used either digital pressure (index finger and thumb of left hand) on the great vessels just at the base of the heart, or a suture method in which a suture was passed beneath the vessels, the two ends of the suture passing through openings in a flat bar which was pressed down on the vessels and which, when the sutures were pulled taut, gripped the vessels between the bar and the threads. This allowed a minimum of handling and the selection of a small area at which it might be desired to make the compression.

We learned that the circulation might in this way be cut off for from two to three minutes, the interval varying with each animal, with satisfactory recovery resulting. In fact, the heart could be made to resume vigorous, normal contractions (by the use of massage, epinephrin and hot saline), even if the circulation were clamped off for eight minutes. When the time interval was three minutes or more, although the heart might be revived and the circulation recovered, the animal was left in a peculiar, decerebrate state. Several such animals died within a few hours or a few days after the operation in marked opisthotonos, with muscular rigidity and twitching. Apparently, when the cerebral circulation is cut off for more than these few minutes, serious, permanent damage results which proves fatal even though the heart is unaffected. The effects of prolonged anoxemia on cells of the nervous system have been carefully studied. Cannon and Cattell,[72] in a recent review of the subject, point out that probably the small pyramidal cells of the cortex withstand anoxemia less well than the remainder of the nervous system, and are probably killed in less than eight minutes, while the cells of the medullary center are more resistant. Certainly, glandular and muscular tissue are far more resistant than nervous tissue elements.

The following experiment illustrates this experience:

EXPERIMENT 1.—Cat 19, weighing 3.3 kg., was etherized by ether vapor blown through an intratracheal catheter. A median incision was made, and the sternum was split. The pericardium was opened, and the heart was delivered. All the blood vessels entering and leaving the heart were clamped for four minutes and ten seconds. During this time, the left ventricle was incised and opened. A papillary muscle was cut. The ventricle was filled with salt solution just before the suture that closed the incision made in the ventricle was tied; the heart beat and respiration ceased during this operation. Just as the pressure on the blood vessels at the base of the heart was released, manual massage was instituted, and after about two minutes spontaneous heart beats occurred. Spontaneous breathing began five minutes after the clamp was removed. The pericardium and chest were closed, and the animal seemed in excellent condition. On the following day it was spastic and was regarded as somewhat decerebrate. Four days after the operation, it was killed.

There were many operative fatalities during these early experiments, chiefly due to the use of an unsatisfactory artificial respiratory apparatus. There were also frequent postoperative deaths from infection resulting in purulent pleuritis and pericarditis. These complications, however, gradually diminished in frequency with experience and improvement in operative technic. For these reasons, it is of no avail to calculate percentages in the early series of experiments as far as mortality is concerned or as to incidence of infection or recoveries. However, after an experience on twenty-three cats in which the closed circuit operation was used, that is, the clamping off of all the vessels at the base of the heart, this procedure was given up. It did not seem that it would be practical to open the heart walls and operate on the valves under direct vision, even if the circulation were clamped during the operation. We arrived at this deduction partly because of the extreme speed with which one always had to work, and the ever-present knowledge that, if this speed were not attained, death would result. No surgeon could conduct a human operation under similar conditions, and since it seemed impossible to lengthen the time interval, we abandoned this method. The experience, however, in reviving hearts that had ceased contracting and in handling the heart proved invaluable to the later work.

B. *Attempts to Create Stenosis by Ligatures, Plication and Radium.*—With this experience in handling the heart as a background, attempts were made to create an experimental stenosis. The experimental production of mitral stenosis has so far proved impossible. It would obviously be of great aid in predicting the beneficial results that might follow the operative reduction of mitral stenosis in human cases, if the condition of stenosis could be experimentally reproduced. The attempts to produce endocarditis in animals by means of micro-organisms [73] have resulted in insufficiency of the mitral valves, due to the inflammatory and vegetative processes which were either rapidly fatal or, if they were not, gave no indication of contracting or constricting the valve in the manner that is so typical of rheumatic mitral stenosis. Therefore, we tried to narrow the mitral valve mechanically by puckering the auriculoventricular ring with a suture placed beneath the coronary vessels, in the region of the mitral valve. In some instances, a wedge-shaped portion of the myocardium was removed with the apex pointing towards the endocardium, and then the gap reapproximated. Neither of these methods proved successful in the production of permanent mitral stenosis. With the former method, it was proved that a valve leaflet might be caught in the suture, thus creating an insufficiency. The animal would recover and show a loud, blowing systolic murmur but no diastolic or presystolic murmur. After a few weeks, however, the systolic mur-

mur would disappear, and when the heart was finally examined, there would be either no evidence of the suture in the auriculoventricular ring or only a trace of it in the musculature. The valves would always appear normal.

The following is an example of one such experiment:

EXPERIMENT 2.—Cat 41, a female, weighing 3.2 kg., was anesthetized with intratracheal ether. A median incision was used splitting the sternum; the pericardium was opened, and a traction suture was inserted at the apex of the heart. One silk suture was taken in the posterior aspect of the upper left ventricle near the auriculoventricular ring. There was good heart action throughout the experiment. The pericardium was closed, and the sternum and skin approximated. The animal left the operating table in excellent condition. No preoperative murmur was heard. Immediately after the operation a loud, blowing, systolic murmur was heard all over the precordium, continuing throughout the entire systole, but no diastolic or presystolic murmur could be heard. Eighteen days later the systolic murmur had markedly diminished in intensity, and seven weeks after the operation it was entirely gone. The animal continued in excellent health for sixteen months, when it was killed for purposes of study. Its weight at death was 3.9 kg. At necropsy, the valves were found to be absolutely normal and there were no signs of the suture. There were a few adhesions between the parietal and visceral pericardium at the apex of the heart.

These methods having failed to produce stenosis, attempts were made after the method proposed by Coryllos, Edwards and Bagg [74] to produce a contracting scar by the use of radium. These investigators used applicators carrying up to from 200 to 400 mc. of radium emanation, which they introduced through the auricular appendix and held in actual contact with the valve edges for some moments. We felt that the injury, in order to simulate better the human disease, should occur in the base of the valves, but we were averse to using large doses of radium close to such important structures as the coronary vessels.

For that reason we adopted one of the clinical methods of radium therapy, and inserted emanation seeds (1 to 8 mc.) by means of a needle into the base of the mitral ring, entering first below the coronary vessels and leaving from four to twelve seeds in this region in each experiment. By this method, we were able to scatter the radium dose pretty widely around the ring and, because of the small amounts used, to leave the seeds in situ.

Dogs were used in these experiments because a larger organ was necessary. There was no difficulty in inserting the radium.[75] Unfortunately, none of the animals have developed definite murmurs. One died from rupture of the coronary vein, due to softening from the effect of radium. Two others have been sacrificed and show interesting dense, firm scars at the site where the seeds were deposited and a definite tendency to retraction of the actual valve segments (Fig. 8). None of the animals have carried the radium longer than three months, and we

are averse to killing any more animals until a longer time has elapsed, for the reason that, in the two dogs killed, the scar was obviously not fully contracted but still in the active, edematous and hyperemic stage of reaction. The chief difficulty encountered in the use of this method is our inability to slide the seeds out actually into the free valve segments. The method, however, holds more promise of success than any other employed in the settling of this question.

Fig. 8.—Two specimens showing reaction to radium placed in the mitral ring of the dog. Arrows indicate areas affected.

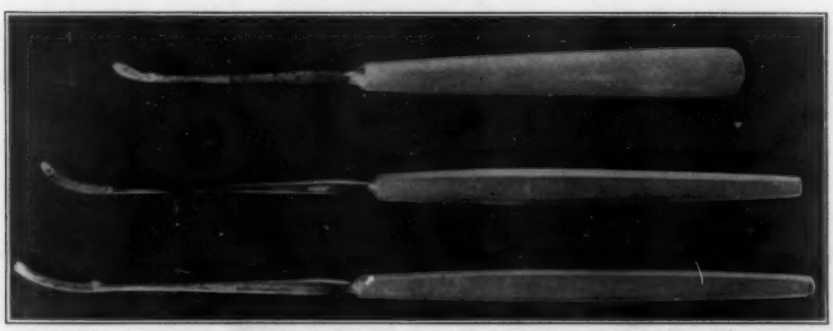

Fig. 9.—The knife-valvulotomes used both in animal experimentation and in Cases 1, 2, and 3.

C. *Operations with Knives Without Interrupting the Circulation.*—Having convinced ourselves that an operation with the circulation closed was not feasible, we began a study of the methods previously used for dividing the valves with small knives on long handles without interrupting the circulation. We selected a knife (Fig. 9) that had a slightly rounded, cutting edge at its top and in which the cutting edge extended

back along the shaft on one side 1 cm. This instrument could, therefore, be easily inserted, and it cut by a motion perpendicular to the attacked valve. The shaft was made tapering toward the cutting end and of such a shape and size as to fill the opening made in the muscle, thus controlling hemorrhage.

The same method of approach was used—the sternum split, the pericardium opened and a suture placed in the apex of the heart for traction, handling and delivery of the organ to the desired position. Having pulled the apex anteriorly and to the right, the knife-valvulotome could then be inserted into the posterior aspect of the left ventricle and directly upwards. After some experience, it was not difficult to find the mitral valve, for the knife could be pushed through to the left auricle where it could be felt. Usually, the knife was passed up the posterior aspect of the ventricle, pushed through the posterior cusp, and then a cut was made outwards towards the free mitral valve edge. If too great pressure were used, the aortic cusp of the mitral valve might be injured, but there was no danger in this maneuver of cutting the wall of the heart chamber. One or two sutures taken at the site of the incision of the left ventricle prevented all bleeding. It was found that no hemorrhage occurred with such an operation, for the body of the knife was so constructed that it effectively blocked the opening it made in the ventricle, and when withdrawn, a suture gave complete closure.

In some experiments, the knife was passed through the mitral orifice and then pulled down in an attempt to cut the aortic flap of the mitral valve. There were, of course, failures to produce a systolic mumur, this latter sign being considered evidence that a defect had been created. One should recall, however, that the tissue that makes up the valve leaflets is thin and in motion, and that it easily escapes injury. In some cases, considerable damage was done to the valves, resulting in loud systolic murmurs, indicating a considerable functional insufficiency. Several animals in which typical murmurs were produced were allowed to live for long periods of time; one for 232 days after such an operation as that described above.

The protocol of this experiment follows:

EXPERIMENT 3.—Cat 32, weighing 5 kg., in which preoperative examination of the heart showed no murmur, was etherized, with the use of an intratracheal catheter. A median incision, splitting the sternum, was made. The pericardium was incised, and a traction suture placed in the apex of the heart. The heart was pulled up by this traction stitch and the valvulotome-knife inserted through the posterior portion of the left ventricle. The innermost end of the knife was directed through the mitral valve and could be seen and felt moving against the wall of the left auricle. The knife was then drawn downwards with the purpose of cutting the aortic cusp of the mitral valve. A resistance was felt in the

act of cutting. The knife was withdrawn, and one silk suture was taken to close the incision in the left ventricle. No hemorrhage occurred. The immediate operation in the heart itself lasted one minute, and the heart and respirations continued essentially normal throughout. A continuous suture was made in the pericardium and the chest closed.

The animal made an excellent postoperative recovery. A moderately loud systolic murmur was heard by four different observers, all over the precordium, but it was most pronounced at the apex of the heart. The animal seemed to be in a perfectly normal condition for about eight months, and at all times manifested a loud, blowing, systolic murmur all over the precordium. Two hundred and thirty-two days after operation, it was killed with chloroform after it had had a bad coryza for two weeks. Necropsy revealed septic bronchitis and pleurisy and the presence of a few pneumonia patches. The heart showed cardiopericardial adhesions at the apex. The mitral valve showed a rent in the aortic cusp; the cut edge was thickened and somewhat granular.

Fig. 10.—The operative defect in the aortic cusp of the mitral valve of Cat 127.

It is quite likely that too great an insufficiency suddenly produced may not be well tolerated. This would be a possible explanation of the death of the following animal so soon after the operation.

EXPERIMENT 4.—Cat 127, weighing 4.4 kg., was etherized by means of an intratracheal tube. A median incision was made, and the sternum was split in the middle. The pericardium was opened and the heart delivered. The apex of the heart was held up with a suture and two sutures were taken in the left ventricle. The knife was introduced between the two sutures and pushed up through the mitral ring into the left auricle. When the knife was drawn down, the aortic cusp of the mitral valve was cut. On withdrawal of the knife, a slight amount of bleeding was observed, and a third stitch, in addition to the original two sutures that had already been placed, was necessary to stop all hemorrhage. The heart at this time was functioning poorly, and the contractions were feeble.

When about 0.75 c.c. of 1/1000 epinephrin solution was injected into the right ventricle, the heart regained its vigor, and the contractions became normal. The pericardium and chest wall were sutured, and the cat left the operating table in a fairly satisfactory condition. An ounce of tap water was given by rectum. After the operation a loud, blowing systolic murmur was heard all over the precordium, transmitted both towards the base of the heart and to the left axilla.

The following day the cat seemed to be in fair shape, except that there was a slight difficulty in breathing. The respirations were 60, and the heart rate was 180. During the following night it died. At necropsy, no evidence of infection in the chest was found. The aortic flap of the mitral valve was cut extensively (Fig. 10), and one of the chordae tendineae was also cut.

Throughout this work, even a few positive results must be regarded as having considerable significance in spite of the many failures. The experiments previously recorded show that, in a cat, the mitral valve can be cut by the insertion of a knife through the left ventricle without direct inspection of the valves. They also proved that the animal could recover good health in spite of the artificial valve defect, and that the incision through the ventricle heals satisfactorily without the formation of endocardial thrombi.

With these experiments on cats as a background, similar work was begun on dogs. In this case we had a larger organ to deal with, and this feature lightened some of the difficulties. At about this stage of our investigations, the operation recorded in Section IX (case 1) was performed, and the patient recovered. We used the identical knife and technic used in the later cat experiments. Following this clinical experience, a large number of dogs were operated on, and it was found that the immediate and remote operative fatalities could be reduced to about 15 per cent.

In the experiments on dogs, it was found more satisfactory to approach the heart through the left chest. The fifth or sixth rib was excised from the sternal attachment to the angle, and then the pericardium was opened. This gave an easy access to the entire heart, and facilitated the proper manipulations. Some of the same types of experiments were carried on with these larger animals as those already described on cats, but in the main the work concerned itself with injuring the valves on the left side of the heart by a ventricular approach.

One series of experiments was undertaken originally with the purpose of cutting the bundle of His, which lies in the upper portion of the interventricular septum; but they also serve to illustrate a type of valve injury that can be produced with a knife-valvulotome by an approach made through the right ventricle, and a cut made through the interventricular septum, thus reaching the aortic valve or even the mitral valve. Such lesions are illustrated by Figures 11 and 12 (Dogs 17, 40,

43, 46 and 85). In Figure 11, *B* (Dog 40), is seen a considerable rent in one of the cusps of the mitral valve and a hole in another portion. This animal survived the operation and continued in good health for 263 days. He was finally killed in a fight that occurred in the animal room. At necropsy the heart appeared enlarged; there was an extensive, adhesive, fibrous pericarditis, the pericardial cavity being entirely obliterated by the process, and a large hole was seen in the mitral valve. There was no evidence that the healing or scarring process had any tendency to restore the rent in the valve to a normal state. The complete protocol follows:

EXPERIMENT 5.—Dog 40, a male, weighing 18 kg., was etherized with intratracheal ether. The left fifth rib was excised from the sternal attachment to the angle, and the pericardium was opened. The anesthetic worked satisfac-

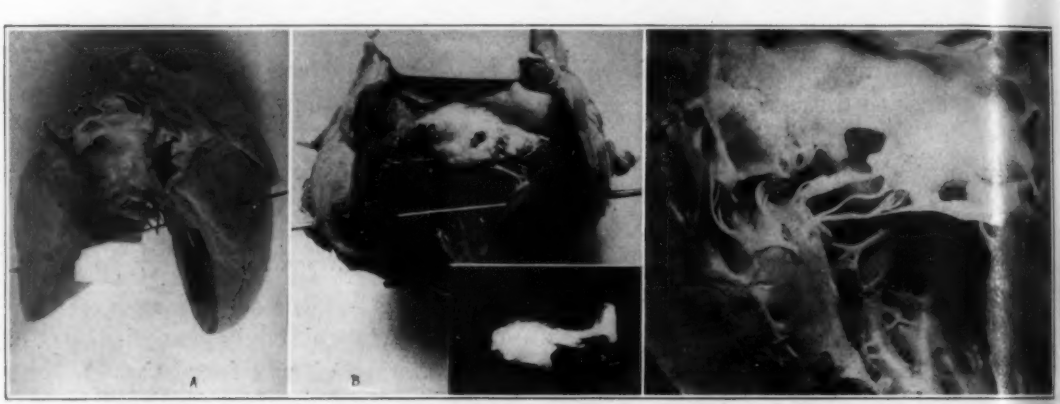

Fig. 11.—Operative lesions in the mitral valves. In *A* the cut lies in the aortic cusp of the mitral valve and in a transverse direction. Animal lived fifty-eight days. The inset in *B* represents a cross section of the valve from the margin of the operative lesion to the base of the mitral ring. Thickening at the edge of the operative defect (to right of inset), giving the appearance of retraction and infolding of the incised margin, may be noted. *C* illustrates the details of the healed margin. Two defects were created at operation. The animal lived 252 days.

torily, and the heart remained small. The knife-valvulotome was plunged through the right ventricle. The left ventricle was entered by cutting the upper portion of the interventricular septum, and then a valve leaflet was cut freely. The knife was quickly withdrawn, and one stitch in the right ventricle sufficed to close the wound. The pericardium and chest wall were closed in the usual way. The animal left the operating table in excellent condition. Examination of the heart directly after the operation revealed a loud systolic murmur not present before the operation. The animal was well and active for 263 days, and on repeated examinations showed a systolic murmur over the precordium. It was finally killed in a fight.

The postmortem examination revealed a normal appearing and healed chest wound. There was no regeneration of bone where the rib was removed. The heart was large, and the hypertrophy involved both ventricles. There were no adhesions between the heart and lungs. The pericardium contained no fluid, the entire cavity being completely obliterated by generalized, extensive, fibrous adhesions. There was a large hole in the aortic cusp of the mitral valve, and two chordae tendineae were cut. Microscopic examination of the cut in the valve showed only dense scar tissue.

EXPERIMENT 6.—A similar result was obtained with Dog 43 (Fig. 11, *C*). This animal also recovered from the operation satisfactorily and was in excellent health for 252 days. He was finally killed, and the heart showed a lesion of the mitral valve. In this case, the left lung was adherent to the chest wall at the site of the wound. Over the surface of the heart were a few small tags, which probably indicated a slight, postoperative pericarditis, which cleared

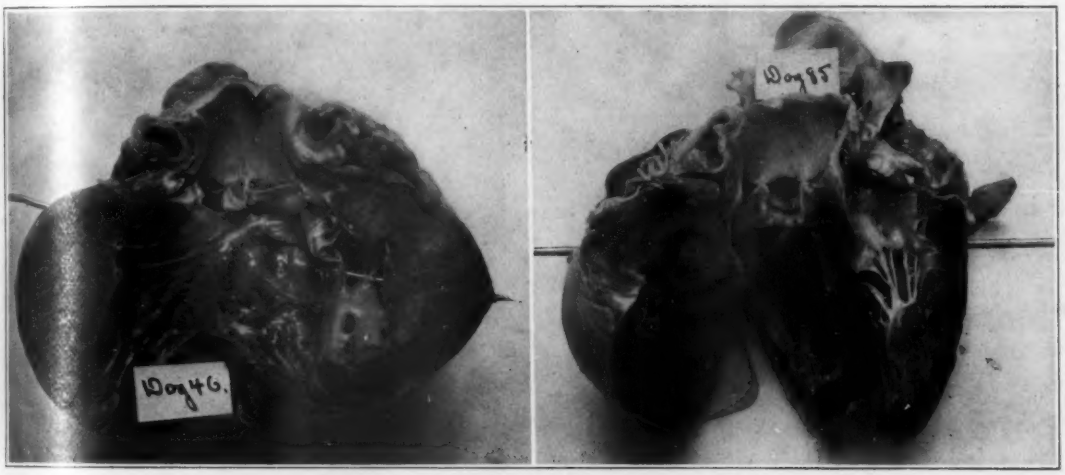


Fig. 12.—Aortic valve lesions. Dog 85 was sacrificed after 23 days.

up. The left lung was adherent to the heart. The mitral valve showed a hole in one portion and an extensive splitting of the free margin in another. The edges of both lesions were curled and thickened, and gave one the impression that the openings would enlarge rather than contract with time. There were no thrombi on the lesions or in the endocardium at the site of incision through the ventricle.

These experiments show that lesions of both the mitral and the aortic valves could be obtained by inserting the valvulotome through the right ventricle and the interventricular septum. Likewise, lesions of these valves may be produced through the left ventricle. Figure 12 shows a rent in one of the cusps of the aortic valve thus obtained. This animal was sacrificed because of distemper twenty-three days after the operation. During life there was heard a systolic and diastolic murmur over the precordium, which indicated an aortic insufficiency. There

were no thrombi either on the valves or in this cavity of the heart. The edges of the valve, according to the photograph, are rumpled and retracted, indicating that the lesion probably would remain permanent.

A study of the necropsy material in these animals showed that

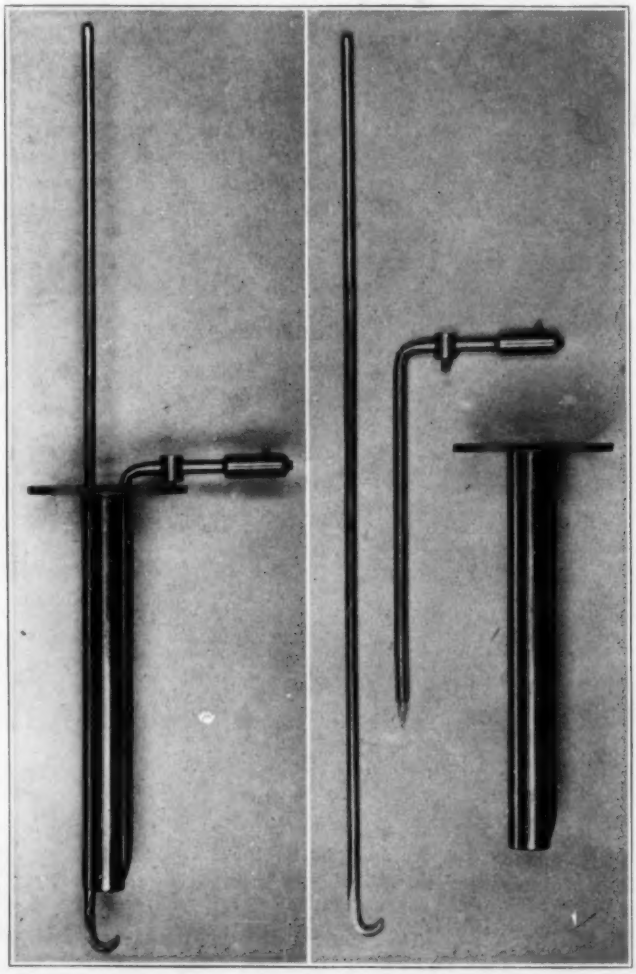

Fig. 13.—Cardioscope designed by Lawrence Rhea and I. C. Walker in 1913. The fact that the lens is somewhat inset made it impossible to have intimate contact between the lens and the object, which perhaps accounts for their failure to secure vision.

valves once cut tend to remain so; that is, the gap remained open, and there seemed to be even a tendency to enlarge as a result of the retraction and curling up of the cut edges. There were no thrombi found on the valves or in the chambers of the heart. even in animals examined a

year or more after the operation. Most of the animals showed pericardial adhesions; generally, these were slight and occurred between the visceral and parietal pericardium at the point of incision of the left ventricle. Sometimes in addition there were adhesions along the line of closure of the pericardium. Occasionally, there were extensive generalized adhesions, but this only resulted when there occurred an infection in the pericardium. Such animals presented a picture not unlike Pick's disease, with adherent pericardium and fluid in the pleural and peritoneal cavities.

D. *Endoscope Experiments.*—These early experiments would be incomplete without some notice of our attempts with an endoscope. When we began these investigations, there was in this laboratory an apparatus (Fig. 13) designed by Drs. Lawrence Rhea and I. C. Walker in 1913. It consisted of a tube carrying in a sheath an electric light, and also a small knife which was constructed for the purpose of cutting the valves of the heart. These investigators never published their attempts, but they have permitted us to state that they were unsuccessful in recovery experiments. In their opinion, the vision afforded was insufficient. We continued investigations with this instrument, using both the ventricular and the auricular appendix approach without success in either seeing valves or in dividing them. Two years later, when Allen and Graham described their similar method, we reinvestigated the whole field of the possible use of such an endoscopic method and had an improved apparatus made. We could never convince ourselves of the benefit to be derived from such visualization, although we are still investigating the possibilities that this method suggests in the hope that the principle of vision may be combined with a more powerful cutting device such as is described in Section V, and which can be inserted through the ventricle. For reasons stated later in full (Section VII), we feel that the auricular appendix approach is neither feasible nor safe.

## DISCUSSION OF EXPERIMENTAL FINDINGS

A series of experiments were conducted on cats and dogs, with the purpose of developing a technic to be used in the surgical treatment of certain heart affections in human beings.

It was learned that the heart could withstand considerable trauma and yet recover normally. The use of hot salt solution applied directly over the exposed heart, manual massage and epinephrin solution was found of great value in restoring proper cardiac contraction.

The entire circulation to and from the heart could be clamped off from two to eight minutes with recovery of a satisfactory circulation; but when the circulation was stopped in this way for more than two or three minutes, although the heart could be made to recover properly,

the animals died in a few hours or a few days in opisthotonos or marked rigidity. It seemed that the cerebral centers, unlike the heart, could not tolerate the standstill in the circulation. Experiments were successfully performed in which the circulation was clamped; during this procedure, the left ventricle was opened and the mitral valve cut under direct inspection. This operation as a general procedure had to be given up because of the damage to the higher nervous centers.

An attempt was made to produce mechanical mitral stenosis by a puckering stitch in the heart in the region of the auriculoventricular ring, and by excising wedge-shaped portions of musculature in this region. These experiments failed because, although insufficiency of the valve lasting a few weeks would result, the valves would always return to a normal condition. Attempts to create stenosis were made by implanting radium emanation seeds about the base of the mitral valve.

An approach to the mitral valve through the left ventricle proved to be successful in cutting valve leaflets without clamping the circulation. Such animals could recover good health and manifest a mitral systolic murmur for many months. When finally examined, the hearts would show that the rent in the valve did not close up, and that there was no tendency to thrombus formation either at the site of the incision through the left ventricle or on the cut valves.

These experiments enabled us to feel that enough had been learned to warrant an attempt to relieve the obstruction in man (Case 1).

## V. EXPERIMENTAL DATA: A NEW METHOD

A. *The Development of a New Instrument.*—The experiments already mentioned indicate that the normal mitral valve of cats and dogs can be sectioned without difficulty by a small knife with a long, slender shaft inserted through the left ventricle, and a mitral regurgitation of variable degree can be produced. The insertion of the knife through the apex of the ventricle is accompanied by no appreciable injury to the heart, and the experiment of cutting the valve and producing mitral regurgitation can be carried out with repeated success. The ease with which this can be done experimentally by means of the valvulotome-knife inserted through the ventricle seemed to outweigh the probable difficulty already anticipated in cutting a stenosed mitral valve in the human, and, at that time, seemed to justify the application of the method to human cases. In the first attempt on a patient, in which the valvulotome-knife was used (Section IX, Case 1), the mitral obstruction seemed to be partially relieved at operation; this was indicated by a change in the heart sounds, an increase in the systolic murmur, and by the improvement in the general health of the patient. In Cases 2 and 3 (Section IX), in which the same method was used, the relief of the obstruction, as shown by the postmortem examination of the heart, was slight but definite, and demonstrated the inadequacy of the simple knife in cutting the stenosed mitral valve to such an extent as to enlarge sufficiently the opening. The difficulty of cutting a stenosed mitral valve with a simple knife was voiced upon *a priori* considerations in a pointed editorial criticism published in the *Lancet* of 1902 immediately following Brunton's communication [5] and also by Tollemer [52] in the *Presse médicale* of that same year.

1. A Consideration of the Physical Properties of the Stenosed Mitral Valve: A consideration of the character of the tissue to be incised emphasizes the importance of certain mechanical principles, the application of which are essential to the successful relieving of the mitral obstruction. The mitral valve undergoes a marked transformation during the disease process. The inflammatory process of the acute stage found along the lines of closure, so delicate in the normal valve, becomes organized, and the margins of the valve become transformed into thickened, irregular, and scarified structures which no longer accurately close. The chordae tendineae are shortened and thickened. Repeated attacks of infection in the natural progress of the disease call forth a marked proliferative and reparative process which adds tissue to the valve, often producing an extraordinary thickness and deformity. Scarification adds to the toughness of the structure, and deposits of calcium give it a rigid and brittle consistency (Fig. 14).

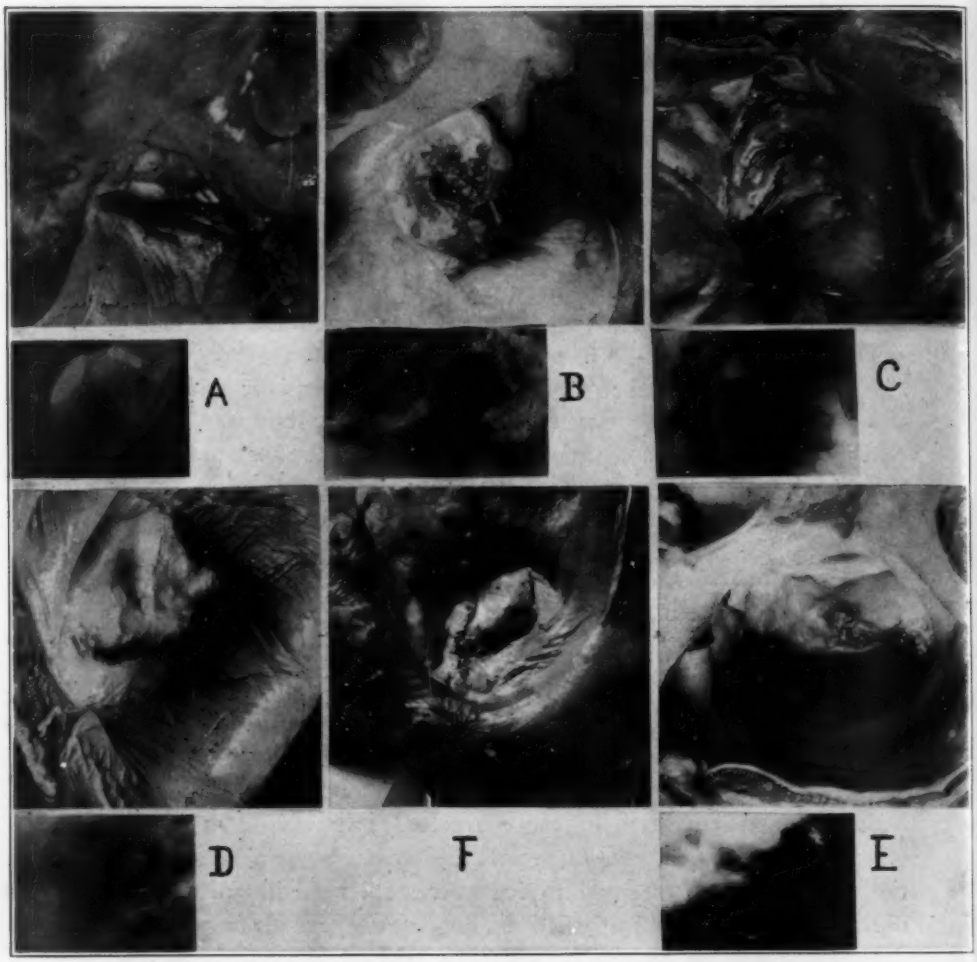


Fig. 14.—Appearance of the orifice in typical cases of mitral stenosis, demonstrating the tendency of the mitral valve in such cases to become thickened and contracted, and to have a slit like aperture. In *A* and *B* synechiae can be seen extending between the cusps. The smaller illustrations are roentgen-ray films of the corresponding stenosed valves. The latter demonstrate the deposition of lime salts and fibrosis.

When the condition of stenotic valves is considered, the degree of obstruction, the thickness of the valve, its fibrous and often calcareous condition, it is apparent that such a valve cannot be sectioned satisfactorily by a force applied to a knife several inches from the cutting edge, without the aid of counter pressure applied to the valve at the point where the section is to be performed. The toughness of such a valve is so marked in certain cases that, indeed, even a pair of scissors with delicate blades is unable to section the valve under direct vision. It seems, furthermore, that a slit-like cut in the markedly thickened valve, on account of the stiffness and the rigidity of the incised margins, may relieve the functional obstruction but little, and that the excision of a piece from such a valve is almost a necessity if the obstruction is to be relieved. It seems essential, therefore, that a powerful cutting device should be employed that will actually excise a piece from the stenosed ring and remove the piece from the blood stream. A valvulotome has been devised which may aid in the accomplishment of this aim.

2. The Cardiovalvulotome:[75a] In the development of the instrument, two guiding principles were accepted from the beginning as a necessity; namely, the principle of cutting by counter pressure, and the application of the force in a line parallel to the axis of the instrument by telescoping the handle. The employment of this latter principle makes the instrument straight and helps greatly in localization when the instrument is in the heart and when the sense of position is so important. In the utilization of the principle of cutting by counter pressure, the simple punch is entirely inadequate in cutting a calcareous valve. To have one cutting edge revolve on the other as it cuts, although capable of cutting the toughest valve, spends too much force in overcoming friction and inertia to be applicable to the sectioning of the stenosed valve in the pulsating heart. The shear, however, is a most efficient cutting principle and affords the ideal mechanical advantage.

In addition to these principles, the desirability of excising a segment from the stenosed valve to relieve the mechanical obstruction was early appreciated. The excision of a piece from the valve, however, necessitates its removal from the blood stream. This is most readily accomplished by shaping the cutting edges into closed-curves, similar to those of a circle or of an ellipse, so that the fragment, as it is excised, becomes encased in the instrument. The cross-section of the shaft of the instrument should be a circle rather than an ellipse, because when it is inserted into the heart the muscle adapts itself poorly to an ellipse if the instrument is rotated, and hemorrhage occurs. To avoid hemorrhage also, the instrument should be of the same diameter throughout the shaft so that when it is in the heart the incised muscle will be under uniform tension.

The valvulotome that has been devised consists of two, close-curved, shearing edges which, by compression of the handle, approximate each

other and accurately override, so that the tissue lying between the cutting edges is excised and encased in the instrument. As the force applied to the handle is released, the cutting edges move apart by virtue of a spring concealed in the handle. The distal portion of the instrument is a bluntly pointed pyramid with a cutting edge on its proximal end which, by telescoping the handle, approximates the opposing cutting edge of the shaft into which it accurately cups. The cutting edges are related as the blades of a shear, and this contributes to making the

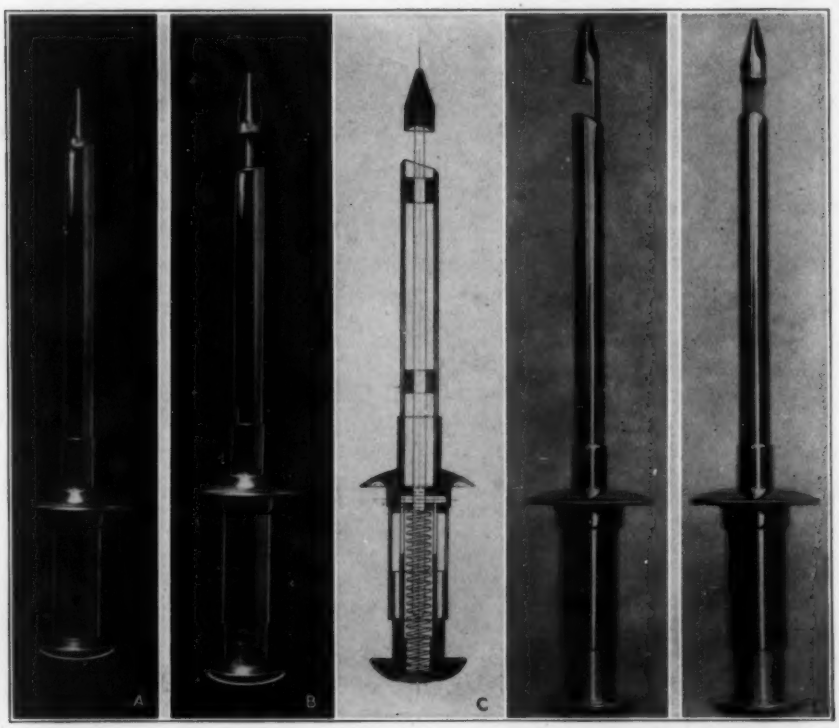


Fig. 15.—The cardiovalvulotome. A, B and C represent the instrument used in Case 4; D and E represent a further development in which the bar carrying the distal portion is eccentric. A represents the instrument in the closed position.

instrument a powerful cutting device. The length of the instrument is 20 cm., and the inside diameter of the shaft is 1 cm. The shape and the relative dimensions are shown in the accompanying photographs and mechanical drawing (Fig. 15).

As a further development of the valvulotome, the bar connecting the distal portion to the shaft has been changed from the midposition to a circumferential position, the length has been made 21 cm., and the inside diameter 0.8 cm. (Fig. 15, *D* and *E*). The eccentric bar has its

advantage in that a relatively larger segment of the stenosed ring can be engaged between the cutting edges and a relatively larger segment thus excised. It has a further advantage in that the excision can be made from either cusp of the mitral valve; and, inasmuch as the aortic cusp usually forms the larger part of the obstructing ring, the excision of a segment from this cusp may be desirable. A line engraved on the handle indicates the position of the cutting edges when the end of the instrument is in the heart.

The length of the shaft is such that the stenosed mitral valve in the human heart can be approached conveniently through the apex of the left ventricle. The diameter of the instrument that can be introduced with safety into the heart through the left ventricle was decided by experiment. Allen and Graham experienced a 50 per cent. mortality accompanying the introduction of a cardioscope through the left ventricle. Elsberg, however, successfully resected one-third of the left ventricle, and our experiments show that the dog can readily tolerate incision, allowing the introduction of the largest valvulotome if it is made near the apex of the left ventricle. This instrument, 1.5 cm. in diameter, although offering no danger if it is inserted through the region of the apex, removed such large segments from the mitral valve of dogs that it produced a regurgitation incompatible with life. The fact that the apex of the ventricle easily tolerates incision is shown in the blood pressure curve (Fig. 29).

Observations made on many stenosed mitral valves [76] indicate that the most markedly stenosed orifice will admit the introduction of the distal portion of the valvulotome having a diameter of 1 cm. The optimum size of the segment to be removed from the stenosed valve in man can only be determined by further experience, but at present the experience afforded by Case 4 indicates that the cutting edges should not be of a diameter greater than 1 cm. Aside from the question of pure dynamics, the increased work suddenly placed on the left ventricle by the section of the valve may constitute an element of necessary consideration. The diameter of the valvulotome, therefore, has been reduced to .8 cm. in order to guard against the removal of a segment sufficiently large to produce an excessive, acute regurgitation. The type of sectioning performed on the stenosed mitral valve by the valvulotome is shown in the accompanying photographs (Fig. 16). The enlargement of the mitral orifice does not appear to be excessive on *a priori* considerations.

B. *Operations with the Valvulotome.*—1. The Operative Method: The following operative procedure was worked out in the laboratory. The dog, having received morphia, is anesthetized with ether administered by a cone. When it is fully relaxed, a properly fitting rubber tube is inserted into the trachea through the larynx. Mechanical respiration

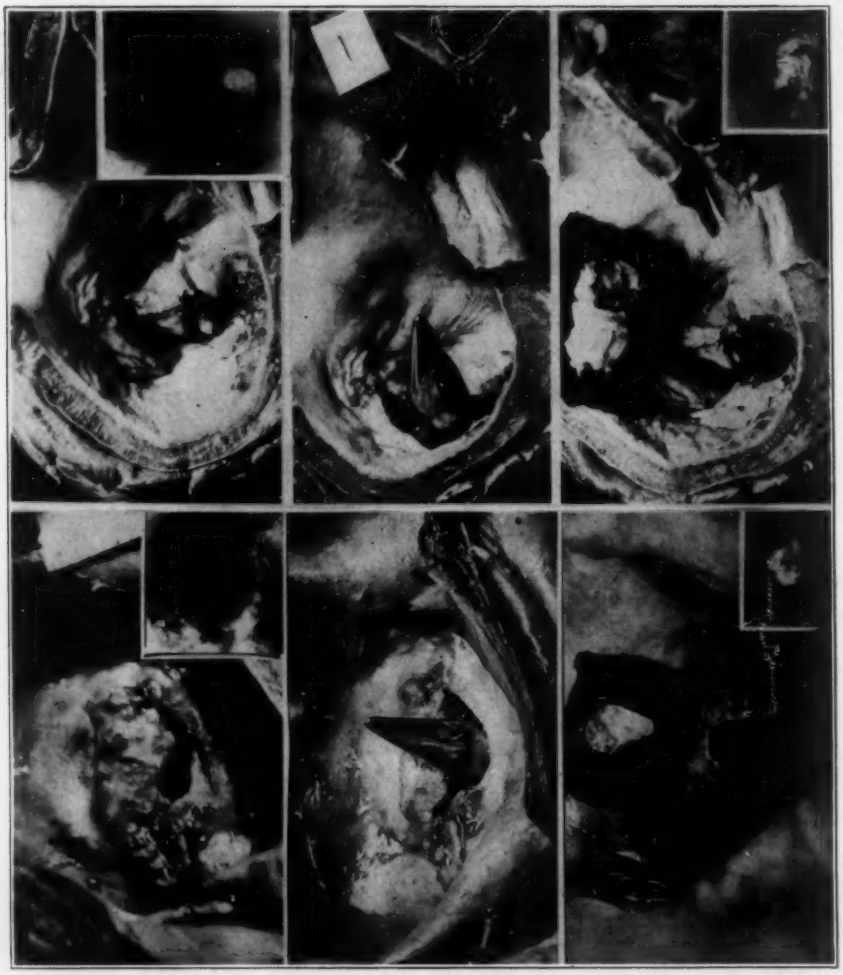

Fig. 16.—Photographs of two hearts showing from left to right: 1, the stenosed mitral orifices; 2, the valvulotome inserted into the orifices, with the stenosed valve engaged between the cutting edges; and 3, the stenosed valves after excision by the valvulotome with the excised segments of the valve. The calcareous nature of the valves is shown by the insets on the left, which are roentgen-ray films of the valves. The insets on the right are roentgen-ray films of the segments excised by the valvulotome and indicate the presence of calcium. The thickness of the excised segments demonstrates the advisability of excision of a piece of valve rather than simple incision, and the presence of calcium indicates the necessity of utilizing the principle of counter-pressure in cutting the valve.

is admirably provided by the Erlanger apparatus [77] (Fig. 17), by means of which air mixed with varying amounts of other vapor is forced intermittently under slight pressure into the trachea. The exhaled air escapes through the space between the intratracheal tube and the trachea,

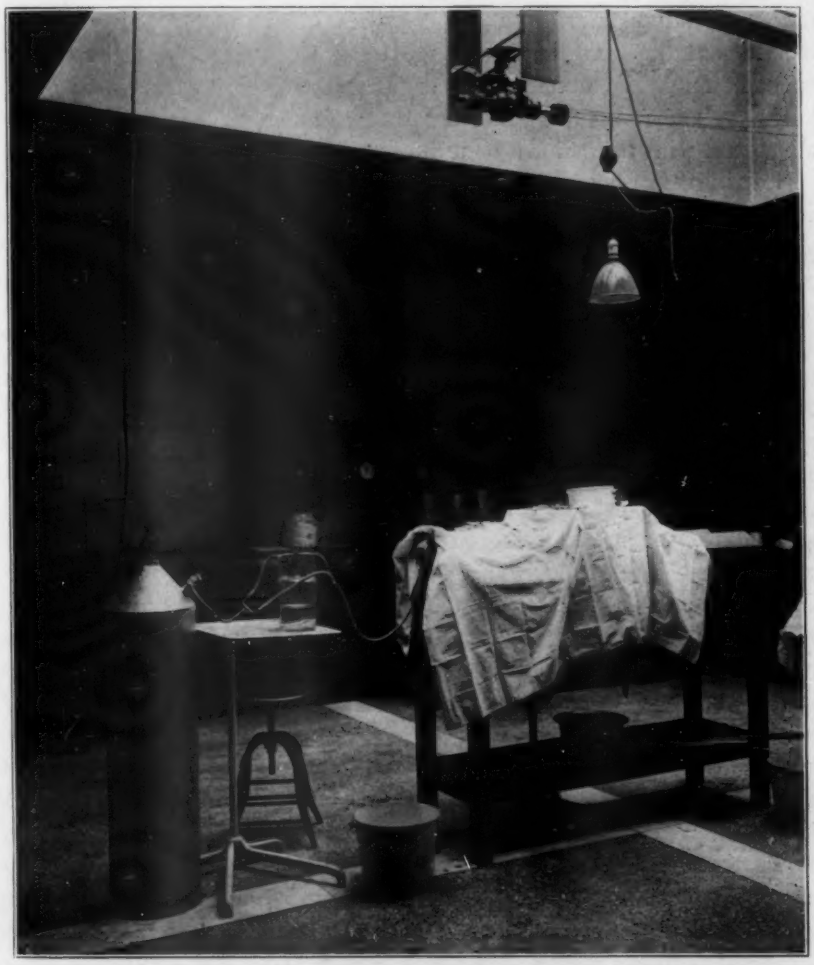

Fig. 17.—Erlanger double chamber cylinder for mechanical respiration. The tube from ceiling carries in compressed air; tube from cylinder containing ether is led to the animal by rubber tubing, one section of which is connected with an ether bottle.

and to obtain the proper degree of aeration of the lungs it is most important that the tube be neither too large nor too small in its relation to the trachea. The dog lies on the right side with the forelegs extended. The thorax is shaved, and the skin is cleansed with soap, water, ether, and alcohol. The sterile drapings are carefully placed, and a most rigid

aseptic technic is observed. An incision made directly over and parallel to the fifth rib is carried through skin, platysma, latissimus dorsi, and sometimes through a small part of the external oblique and the pectoralis minor muscles to the periosteum of the rib. The fifth rib is removed subperiosteally from the costochondral articulation laterally about 4 inches (10.16 cm.). The pleura is incised and the margins of the wound are retracted. As the chest is opened, it may be necessary to regulate the degree of aeration of the lungs.[78] The lungs are protected by two or three thin pads of cotton moistened with normal salt solution at a temperature of 105 F., and the pericardium is exposed. It is incised to the right of the left phrenic nerve, and the heart is exposed from base to apex. Any bleeding point in the pericardium should be ligated. Retraction of the pericardium is provided by traction sutures. It is of the greatest importance to avoid trauma to the pericardium and to the epicardium, not because of a disturbance to the mechanism of the heart, but because of the marked sensitivity of the heart and the pericardium to the subsequent formation of adhesions. The heart is displaced from the pericardial cavity by a spatula, and a suture placed in the apex provides for traction and handling of the heart. A region devoid of coronary vessels as near the apex as possible and away from the base of the anterior papillary muscle is selected for the introduction of the valvulotome. Two sutures of silk taking a deep bite of muscle are placed in the area selected for the insertion of the valvulotome so that the four threads represent on the surface of the heart a square of 1 cm. These are the control sutures, and they are of importance in the control of hemorrhage. A small scalpel, placed between the control sutures so as to bisect the hypothetical square, cuts completely through the ventricular wall in a plane slightly oblique to the surface of the heart. The oblique incision has its advantage in that it tends to close without bleeding, but its disadvantage in that a greater depth of muscle is incised. If there is any bleeding, and experience has shown that there need be no bleeding, the control sutures, which have been crossed, are made slightly taut. The valvulotome, after having been filled with salt solution to displace the contained air, is introduced through the incision into the cavity of the left ventricle, and is then allowed to open. The cutting edges are introduced into the mitral ring, the position being determined accurately by the index finger of the left hand as it feels the end of the instrument through the invaginated wall of the left auricle. The mitral valve leaflets now lie between the cutting edges and, by telescoping the handle, are excised and encased in the instrument, which is then removed from the heart. Slight traction on the control sutures prevents any bleeding as the instrument is slowly withdrawn, and the wound is closed with one or two silk sutures (Figs. 18, 19, 20). The control sutures and the apex suture are

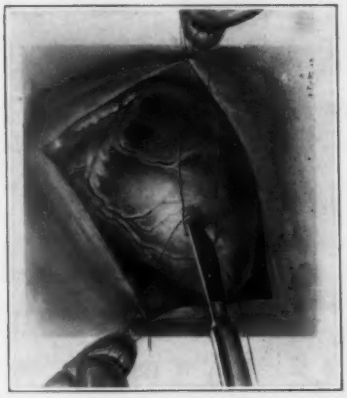

Fig. 18.—Stages in the operative procedure in animals; this also applies to human cases. The control sutures are in place and the incision is being made with a knife.

Fig. 19.—Insertion of the cardiovalvulotome; the inset shows the distal end of the instrument and a drawing of the actual segments of the mitral valve of Dog 108.

Fig. 20.—The method of closure. Lembert sutures being inserted while the control sutures approximate the wound.

removed, and the heart is replaced in the pericardial cavity. The pericardium is sutured, and before the pleura is completely closed, the lungs are expanded to expel the air from the chest, and the wound is sutured in layers.

2. Protocols: As already indicated, the valvulotome has undergone several improvements in its construction from the original model to the models just described, without, however, involving any change in the fundamental principles.

The length and the diameter of the shaft, the shape of the distal portion, the degree of the shear, etc., represent details which were suggested naturally by its application on animals (dogs and goats). Likewise, many details in the operative method were improved by experiment, and often fatal experiment, on the dog. Such details included the proper spacing and depth of the control sutures, the amount of traction that can be safely applied to the sutures without tearing the muscle, the avoidance of air embolism in the coronary vessels, the handling of the heart and, finally, the establishment of those surgical reflexes so necessary to the performance of cardiac surgery. These experiments, numerous as they appear on reflection, are not included in the series given below.

The operative technic, as it was finally evolved and as has been described above, was not fully established before the series of experiments reported here was begun. Thus, the advisability of avoiding the base of the anterior papillary muscle in making the incision in the ventricle was not appreciated until it resulted in two fatalities included in the series, and undue traction on one of the control sutures tore the muscle and produced death by hemorrhage. This series includes the last thirty consecutive operations on the dog, in which an attempt was made to excise a segment of the mitral valve, with recovery of the animal. Of these animals, twenty-four survived the operative procedure, almost all of which were given a well marked mitral insufficiency.

Inasmuch as it seems unnecessary to discuss in detail each of the thirty experiments, the protocols of the first fourteen consecutive experiments are given to illustrate the incidents characteristic of the operation itself and of the postoperative course. The accompanying photograph shows the segments of the mitral valves removed at operation (Fig. 21).

PROTOCOLS

EXPERIMENT 7.—Dog 97, a red and white male setter, weighing 20 kg., Jan. 17, 1924, was given one-fourth grain (0.0162 gm.) morphin; the heart rate was 92, and there was no murmur; blood pressure taken by means of a cuff sphygmomanometer applied over the upper thigh was: systolic, 230; diastolic, 60. According to the roentgen rays, the transverse diameter of the heart was 6.8 cm.

*Operation.*—Intratracheal ether was administered with the Erlanger apparatus. The dog was on its right side with the left foreleg extended, and the skin was shaved and cleansed with soap and water, ether and alcohol. An incision was made over the fifth rib extending from the costochondral articulation laterally about 5 inches (12.7 cm.) and carried down to the rib, which was resected subperiosteally. The pleura was opened, and the expansion of the lungs was carefully regulated. Thin pads of cotton moistened with a normal salt solution at a temperature of 105 F. were placed to protect the lungs and to expose the pericardium. The left phrenic nerve was avoided in opening the pericardium. The heart was displaced from the pericardial cavity by a spatula; a suture, to be used in subsequent manipulation of the heart, was placed in the apex of the left ventricle. An area of the left ventricle, free from coronary vessels, was selected for the insertion of the valvulotome. The control sutures were placed in this area as described above, crossed, and held without tension

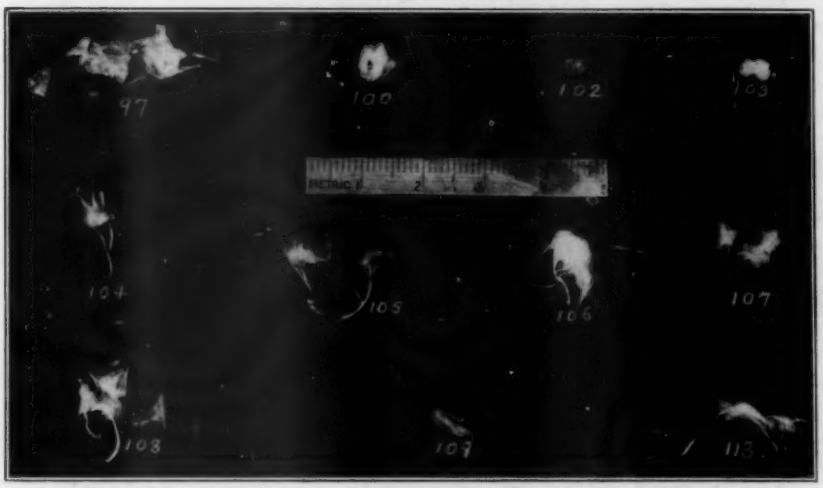


Fig. 21.—Segments of the mitral valve removed at operation. Dog 97 died forty-one days after operation. Dog 100 was killed for photograph of heart 107 days after operation. Dog 102 is living ninety-eight days after operation. Dog 103 died six hours after operation. Dog 104 is living, 106 days after operation. Dog 105 had an operative death. Dog 106 died thirty-three days after operation. Dog 107 is living ninety-one days after operation. Dog 108 died four days after operation. Dog 109 is living eighty-four days after operation. Dog 113 is living seventy-four days after operation.

by the assistant. The operator, steadying the heart by the apex suture, made an incision in the ventricle between the control sutures. A small spurt of blood occurred, but this was controlled by slight traction on the sutures. The valvulotome was inserted; the control sutures were dropped; there was no bleeding; and the heart was steadied by the assistant holding the apex suture. The operator placed the cutting edges of the instrument in the mitral ring, the position being determined easily by feeling the end of the instrument through the invaginated wall of the auricle by the index finger of the left hand. The valve was cut, and, as the valvulotome was removed from the heart, the assis-

tant dropped the apex suture and exerted slight traction on the control sutures. There was no bleeding. The incision in the ventricle was closed with one Lembert suture of silk. The heart action remained good. The pericardial cavity, after having been cleansed by cotton pledgets moistened with normal salt solution, was closed with a continuous silk suture. The cotton pads were removed. There was no blood in the chest. After expelling the air from the chest, the pleura was closed by suturing each layer of the periosteum. Two continuous sutures approximated the muscles, and a continuous subcutaneous suture in the platysma closed the skin.

A large piece of the mitral valve containing several chordae tendineae had been removed (Fig. 21). A loud systolic murmur was heard. The pulse rate was 200; blood pressure, systolic 200, and diastolic 120; according to the roentgen ray, the transverse diameter of the heart was 7.2 cm.

Six hours after operation there was a loud systolic murmur; the pulse rate was 180, and the dog lay quietly in the cage.

January 18: Pulse rate, 176; the dog stood up as the door to the cage was opened.

January 19: Nose cool; pulse rate, 168; the dog walked from his cage.

January 21: Pulse, 162; according to the roentgen ray, the transverse diameter of the heart was 7.2 cm.

January 24: Pulse rate, 174; loud systolic murmur; transverse diameter of heart (roentgen ray) 7.3 cm.

January 30: Pulse rate, 176; the dog had a slight cough, and the respirations seemed to be a little rapid; the transverse diameter of the heart (roentgen ray) was 7.6 cm.

February 12: Pulse, 188; loud systolic murmur; the general condition of the dog was good; transverse diameter of the heart (roentgen ray) 7.8 cm.

February 18: Under morphin the pulse rate was 138, and the blood pressure was systolic, 130, and diastolic 94; the transverse diameter of the heart (roentgen ray), 8.2 cm.

February 27: The dog was found dead in the stall. The mouth and nose were filled with pink froth. No fluid was present in the chest. The lungs were not adherent to the chest, but in one small area there was an adhesion to the pericardium. The lungs were distended and boggy, and when they were cut with a knife, fluid exuded from the incised tissue. There were no signs of pneumonia. The pericardium was adherent to the heart over the incision in the ventricle, but elsewhere it was free. The pericardial fluid was small in amount and clear. The heart was moderately enlarged, weighing 242 gm. The left auricle was capacious and definitely enlarged. The tricuspid ring was dilated, making the tricuspid valve insufficient. The scar in the left ventricle was firmly healed and without thrombus formation. A large leak existed in the mitral valve produced by the removal of a segment of the aortic cusp of the mitral valve and the section of several chordae tendineae (Fig. 22). There was no fluid in the abdomen. The liver was congested, weighing 753 gm. The spleen and kidneys showed no congestion.

The microscopic examination of the lungs revealed marked edema and congestion. There was no pneumonia. The liver was congested. The spleen and kidneys were normal.

Death was attributed to mitral insufficiency, with relative tricuspid insufficiency and congestion of the lungs and liver.

EXPERIMENT 8.—Dog 98, a black and brown male mongrel, weighing 18.8 kg., Jan. 18, 1924, was given one-fourth grain (0.0162 gm.) morphin; the

heart rate was 122, and there was no murmur. The blood pressure taken by a cuff sphygmomanometer was: systolic 130; diastolic could not be determined; the transverse diameter of the heart (roentgen ray) was 7.3 cm.

*Operation.*—The procedure described above was carried out. The fifth rib was resected. The apex and the control sutures were placed. The incision was made in the ventricle, and the valvulotome was inserted. The valvulotome, after its withdrawal from the heart, did not contain a piece of the valve. It was reinserted, and a small segment of the valve was removed. Hemostasis was perfect. The heart action was good; the heart was not dilated, and the color was good. Two Lembert sutures of silk closed the incision in the heart;

Fig. 22.—Heart of Dog 97, illustrating defect in aortic cusp of the mitral valve; the animal died forty-one days after operation from pulmonary edema.

the usual closure was made. The condition after the operation was good. There was a faint systolic murmur, and the pulse was 155. The blood pressure was: systolic, 180; diastolic, 104. The transverse diameter of the heart (roentgen ray) was 7.7 cm.

January 19: Soft systolic murmur; pulse rate, 155; the dog stood up in its cage.

January 21: Murmur persisted; pulse rate, 150. The blood pressure was: systolic, 170; diastolic, 114; the transverse diameter of the heart (roentgen ray) was 8.2 cm.; the nose cool; the condition was apparently satisfactory.

January 30: Pulse rate, 148; short systolic murmur; the dog was active and strong.

February 13: The transverse diameter of the heart (roentgen ray) was 7.8 cm.

March 21: The dog, having become one of the dominating members of his group, was killed in combat with other dogs.

The chest wound was firmly healed, the rib not regenerated. There were no signs of cardiac decompensation, and no fluid was present in the chest. The left lung was adherent for about 1 cm. to the scar in the chest. The lungs were normal, and there were no adhesions between lung and pericardium. There was no fluid in the pericardial cavity. The pericardium was firmly adherent to the heart at the region of the incision in the ventricle; elsewhere, it was free. The heart did not seem to be enlarged. The incision in the muscle was firmly healed without thrombus formation. There was a small lesion of the anterior portion of the aortic cusp of the mitral valve, producing a slight insufficiency. The valve margin was thickened and scarified. One chorda tendinea was cut. There were no thrombi on the healed margins. The abdomen was negative; there was no free fluid, and the liver was not congested.

EXPERIMENT 9.—Dog 100, a yellow and white male collie, weighing 13.6 kg., was given, Jan. 23, 1924, one-half grain (0.0324 gm.) morphin; the heart rate was 72, and there was no murmur; the transverse diameter of the heart (roentgen ray) was 7.3 cm.

*Operation.*—The usual procedure was employed. The fifth rib was resected, and the apex and control sutures placed. Incision was made in the ventricle. As the valvulotome was being inserted, traction on the apex suture tore the muscle and resulted in some bleeding, which was controlled by the placement of another suture in the apex. The valvulotome was reinserted, and the mitral valve was sectioned. The segment of the valve is shown in Figure 21. The incision was closed with one mattress suture, and the usual closure was made. There was a loud systolic murmur; the pulse rate was 176, and the transverse diameter of the heart (roentgen ray) was 7.5 cm.

January 24: Loud systolic murmur; pulse rate, 170.

January 25: Condition good; dog stood in its cage.

January 30: Loud systolic murmur; pulse rate, 160; good condition.

February 13: Pulse rate, 144; the transverse diameter of the heart (roentgen ray), 7.8 cm.

February 18: One-sixth grain (0.0108 gm.) morphin; blood pressure, systolic, 172, and diastolic, 94; rate, 120; loud systolic murmur; diameter of heart (roentgen ray) 8.2 cm.

March 25: Pulse rate, 120; diameter of heart (roentgen ray) 8 cm.

April 24: General condition excellent; weight of dog, 17 kg., an increase of 3.4 kg. over preoperative weight. Pulse rate, 120; diameter of the heart (roentgen ray), 7.8 cm. The heart had responded to the mitral insufficiency by an increase in rate and slight hypertrophy; the roentgenograms taken throughout the postoperative period showed a prominence in the region of the left auricle. The immediate postoperative increase in size was due to dilatation, and this was followed by hypertrophy. The dog was killed May 9, in order that a photograph (Fig. 23) might be obtained for publication.

EXPERIMENT 10.—Dog 101, a yellow and white male collie, weighing 13.6 kg., was given Jan. 24, 1924, one-half grain (0.0324 gm.) morphin; the heart rate was 96, and there was no murmur. The transverse diameter of the heart (roentgen ray) was 6.2 cm.

*Operation.*—The usual procedure was followed. The fifth rib was resected; apex and control sutures were placed. The ventricle was incised with the scalpel, and the valvulotome inserted. Two attempts were made to cut the mitral valve. A small piece of tissue was removed consisting of endocardium, but the presence of mitral valve is questionable. The tissue was lost at operation. One mattress suture closed the wound. There was no bleeding, and the usual closure was made. The condition was excellent; the pulse rate was 130. There was a questionable short murmur following the first sound; the transverse diameter of the heart (roentgen ray) was 6.8 cm.

January 25: There was a faintly heard, short systolic murmur; pulse rate, 108; the dog stood up in its cage.

January 30: Short systolic murmur; good condition.

February 13: The presence of a definite murmur was questionable; pulse rate, 128; the transverse diameter of the heart (roentgen ray) was 6.3 cm.

Fig. 23.—Heart of Dog 100; a large segment has been removed from the free edge of the posterolateral cusp. Pointer indicates scar in ventricle where instrument had been inserted.

February 18; one-sixth grain (0.0108 gm.) morphin was given; pulse rate, 80. The blood pressure was: systolic, 142; diastolic, 80; questionable murmur; diameter of heart (roentgen ray), 6.5 cm.

March 25: Pulse rate, 102; blood pressure, systolic, 160, and diastolic, 70; diameter of heart (roentgen ray) 7 cm.

April 24: Roentgen ray, shadow of heart, 7.2 cm.; the first sound was roughened. The dog probably had a slight mitral regurgitation. The heart, as indicated by roentgen-ray shadows, was increasing in size. The enlargement immediately following the operation was due to dilatation of the heart, but the subsequent enlargement may have been due to hypertrophy. The general condition was good. The dog had gained 2 kg. since the operation.

EXPERIMENT 11.—Dog 102, a yellow and white male mongrel, weighing 10 kg., Jan. 28, 1924, was given one-sixth grain (0.0108 gm.) morphin; the heart

rate was 84, and there was no murmur. The blood pressure was: systolic, 160; diastolic, 104; the transverse diameter of the heart (roentgen ray) was 5.8 cm.

*Operation.*—The usual procedure was followed. Apex and control sutures were placed, one of the latter piercing a small coronary artery, which caused a little bleeding. The incision was made, the valvulotome inserted and a small segment of the mitral valve removed (Fig. 21). The wound was closed with three Lembert sutures. The heart action was regular and forceful. The usual closure was made. There was a soft, systolic murmur; the pulse rate was 168, and the roentgenogram of the heart measured 5.8 cm.

January 29: The dog lay in its cage; nose cool; systolic murmur; pulse rate, 150.

January 30: Pulse rate, 144, regular; condition good; dog stood up in its cage.

February 13: Well defined systolic murmur; pulse rate, 128; roentgenogram of heart measured 6 cm.; the dog appeared to be losing weight, and there was a slight discharge from the nose.

February 18: The dog developed distemper and was becoming emaciated; it was killed with ether. There was a small infected pressure sore on the left foreleg, and a discharge from the nose.

*Necropsy.*—The incision in the chest was firmly healed. The left lung was adherent to the scar in the chest. No fluid was present in the thorax. A number of small, grayish, emphysematous areas were scattered over each lung. In the posterior part of the middle and lower right lobes was an area of consolidation, a microscopic section of which showed may red blood cells and considerable edema in the alveolar spaces. The lung was adherent to the suture line of the pericardium, and the latter was firmly adherent to the heart at the scar of the incision. There was no pericardial fluid. The scar in the ventricle was firmly healed without thrombus formation. There was a small lesion at the anterior junction of the mitral valve leaflets with several sectioned chordae tendineae, sufficient to produce a regurgitation. The liver and spleen were normal, and no fluid was present in the abdomen.

EXPERIMENT 12.—Dog 103, a yellow and white female hound, weighing 15 kg., was given, Jan. 29, 1924, one-fourth grain (0.0162 gm.) morphin; the heart rate was 104, and there was no murmur. The blood pressure was: systolic, 160; diastolic, 82; the transverse diameter of the heart (roentgen ray) was 6.2 cm.

*Operation.*—The usual procedure was followed. The segment of the mitral valve removed is shown in Figure 21. The incision in the ventricle was closed with three Lembert sutures. There was no bleeding, and the usual closure was made. The condition of the dog was excellent; there was a soft, systolic murmur; heart rate, 180; roentgen-ray shadow of the heart, 6.4 cm.

Six hours after operation the dog was found dead. A complete necropsy revealed no concomitant cause of death. There was no heart tamponade, and no embolus was present in the lungs or in the brain. The lungs were normal; there was no edema, and no consolidation. The abdomen was normal. There was a lesion of the aortic cusp of the mitral valve producing a moderate regurgitation. A lesion of this size is ordinarily well tolerated by most dogs, but it has been noticed that the hound is, on the average, less viable than other species of dogs.

EXPERIMENT 13.—Dog 104, a yellow, white and gray male collie, weighing 8.3 kg., Jan. 30, 1924, was given one-sixth grain (0.0108 gm.) morphin; the heart rate was 80, and there was no murmur. The blood pressure was: systolic, 170; diastolic, 90; the transverse diameter of the heart (roentgen ray) was 6 cm.

*Operation.*—The usual procedure was followed. The fifth rib was resected subperiosteally. The heart was displaced from the pericardial cavity by a spatula, and a suture was placed in the apex of the left ventricle for subsequent manipulation of the heart. Two control sutures were placed in the left ventricle in an area devoid of coronary vessels. The sutures were crossed and held without tension by the assistant. An incision oblique to the surface of the heart was made between the control sutures. There was no bleeding. The valvulotome was inserted into the ventricle, and the cutting edges were placed in the mitral ring, the position being determined by feeling the end of the instrument through the invaginated wall of the left auricle. The valve was cut, and as the instrument was withdrawn from the heart, slight traction was made on the control sutures by the assistant. There was no bleeding. The incision in the ventricle was closed with two Lembert sutures of silk on French needles. The control and the apex sutures were removed. The heart was slightly dilated, regular and not cyanotic. The pericardium was closed with two interrupted sutures. The wound was closed in layers.

A large segment of one of the mitral valve cusps, including several chordae tendineae, had been removed by the valvulotome (Fig. 21). There was a very loud murmur extending throughout systole, and the pulse rate was 180 per minute. The transverse diameter of the heart as shown by the roentgen ray was 6 cm.

Seven hours after the operation, the heart rate was 200, with a loud systolic murmur; the animal appeared bright.

January 31: Heart rate, 178; loud systolic murmur; the dog's nose was cool.

February 1: Heart rate, 160; the dog walked from the cage, and took food and water.

February 5: Heart rate, 144; good condition.

February 13: Heart rate, 132; loud systolic murmur; transverse diameter of the heart as shown by the roentgen ray, 5.8 cm.

February 18: One-sixth grain (0.0108 gm.) morphin given; heart rate, 100; blood pressure, systolic, 100, and diastolic 40; loud systolic murmur; transverse diameter of the heart as shown by the roentgen ray, 6.2 cm.

March 28: Loud murmur; pulse rate, 160; blood pressure, systolic, 120, and diastolic, 60; transverse diameter of the heart (roentgen ray) 6.7 cm.

April 24: Diameter of heart (roentgen ray), 6.9 cm.; the heart was becoming hypertrophied. The general condition of the dog was good, and since operation there had been a gain in weight of 2.3 kg.

EXPERIMENT 14.—Dog 105, a white and tan, male mongrel setter, weighing 20 kg., a very old dog, Jan. 21, 1924, was given one-fourth grain (0.0162 gm.) morphin; the heart rate was 88, and there was no murmur.

*Operation.*—The usual procedure was followed. As the apex suture was placed, it was noticed that the heart was very sensitive; the placing of the apex suture caused it to beat very rapidly, and when the finger was placed in the mitral ring through the invaginated auricle the heart beat rapidly, became dilated and remained dilated for several minutes. The control sutures were

then placed, and the incision in the ventricle made. The heart again beat very rapidly. The valvulotome was inserted, and a segment of the mitral valve was excised and removed with the instrument. There was no bleeding. The heart beat feebly, and as the wound was being sutured it stopped beating. Epinephrin failed to bring about recovery.

Examination of the heart showed a lesion at the junction of the aortic and posterior leaflets of the mitral valve involving both leaflets and several chordae tendineae. Death may have been due to the excessive regurgitation, although the sensitivity of the heart to the operative procedure may have been a factor (Fig. 21).

EXPERIMENT 15.—Dog 106, a male coach dog, weighing 18.6 kg., Feb. 1, 1924, was given one-third grain (0.0216 gm.) morphin; the heart rate was 90, and there was no murmur. The blood pressure, taken by cuff sphygmomanometer was: systolic, 210; diastolic, 156; the transverse diameter of the heart (roentgen ray) was 7 cm.

*Operation.*—The usual procedure was followed, and the fifth rib resected; apex and control sutures were placed; the incision was made in the left ventricle with a scalpel, and the valvulotome inserted; a large segment of the mitral valve with several chordae tendineae was removed by the instrument; the incision was closed with three Lembert sutures, and there was no bleeding. On the removal of the segment of valve, the heart action became rather feeble and the heart considerably enlarged. The pericardium was sutured. Usual closure. The heart rate was 150, and a very loud murmur extended throughout systole; the transverse diameter of the heart (roentgen ray) was 7.2 cm. The valve removed is shown in Figure 21. If recovery with this lesion should occur, it would be an excellent example of the endurance of the heart.

February 2: The dog lay in the cage; heart rate, 150; loud murmur.

February 3: The dog stood up in its cage; condition good.

February 6: Heart rate, 114; loud systolic murmur; condition good.

February 13: Loud, blowing, systolic murmur persisted; heart rate, 168; transverse diameter of the heart (roentgen ray), 8.5 cm.

February 18: One-fourth grain (0.0162 gm.) morphin; blood pressure, systolic, 142, and diastolic, 80; heart rate, 164; loud systolic murmur; transverse diameter of the heart (roentgen ray), 8.2 cm.; the dog seemed to be losing weight.

February 20: The transverse diameter of the heart (roentgen ray) was 8.2 cm.

March 5: The dog was found dead. He had been losing a little weight, and did not seem to be normally active throughout the postoperative period.

*Necropsy.*—Pink froth was found in the trachea. The chest wound was well healed and the lungs were not adherent to the wound. There was about 20 c.c. of bloody fluid in the left chest, and no fluid in the right chest. The lungs were voluminous and edematous and everywhere wet and water-logged. Considerable fluid oozed from the freshly cut surface of the lung. A small adhesion existed between the pericardium and the lung. No fluid was present in the pericardial cavity; there was a generalized pericarditis, the adhesions being easily separated everywhere with the fingers except over the scar in the ventricle. The heart seemed to be somewhat enlarged; its weight was 175 gm. The scar in the ventricle was firm, and there was no thrombus. A large defect existed in the posterolateral cusp of the mitral valve (Fig. 24). The margin

of the valve healed without thrombus formation. The left auricle was capacious. The abdomen did not contain free fluid; the liver was enlarged and congested.

*Microscopic Examination.*—The lungs showed much fluid in the alveolar spaces; the liver showed beginning central necrosis with congestion, and the kidneys were slightly congested.

EXPERIMENT 16.—Dog 107, a white and tan collie, female, weighing 13.5 kg., Feb. 4, 1924, was given one-sixth grain (0.0108 gm.) morphin; the heart rate was 96, and there was no murmur. The blood pressure was: systolic, 170; diastolic, 90; the transverse diameter of the heart (roentgen ray) was 6.7 cm.

Fig. 24.—Heart of Dog 106; large operative lesion in posterolateral cusp extending from valve edge to base of ring; scar healed; no thrombi.

*Operation.*—The fifth rib was resected. The pleura was opened and the aeration of the lungs properly regulated. The lungs were protected with thin pads of cotton moistened with salt solution. The pericardium was incised and retracted. The apex and the control sutures were placed. The incision was made in the ventricle, and the volvulotome was introduced. The mitral valve was cut. The incision in the ventricle was closed with two sutures. There was no bleeding. The heart was slightly dilated, but beat regularly and was of good color. The pericardium was sutured. The pads of cotton were removed, and the chest was closed in the usual manner.

A segment of each mitral valve cusp was removed by the valvulotome, producing a marked regurgitation (Fig. 21). The first sound was very soft and scarcely audible, and a murmur was heard throughout systole. The heart rate was 180; the transverse diameter of the heart (roentgen ray) was 7.7 cm.

Eight hours after the operation, the heart rate was 160, and there was a murmur throughout systole. The dog stood during auscultation of the heart.

February 6: Heart rate, 110; loud systolic murmur; the dog was active, and apparently in a good condition.

February 13: Heart rate, 130; systolic murmur; a pleural friction rub was heard over the incision and the transverse diameter of the heart (roentgen ray) was 7.2 cm. (Fig. 28).

February 18: One-sixth grain (0.0108 gm.) morphin was given: heart rate, 152; the blood pressure was: systolic, 122, and diastolic, 72; loud systolic murmur; transverse diameter of the heart (roentgen ray), 7.4 cm.

March 28: The transverse diameter of the heart (roentgen ray) was 7.7 cm.

April 2: The dog was in good condition; the transverse diameter of the heart (roentgen ray), 7.4 cm.; present weight, 16 kg.

EXPERIMENT 17.—Dog 108, a black and white male collie, weighing 16.6 kg., Feb. 6, 1924, was given one-fourth grain (0.0162 gm.) morphin; the heart rate was 120, and there was no murmur. The blood pressure was: systolic, 164; diastolic, 104; the transverse diameter of the heart (roentgen ray) was 6.8 cm.

*Operation.*—The fifth rib was removed subperiosteally. The heart was displaced from the pericardial cavity by a spatula, and a suture was placed in the apex of the left ventricle for subsequent manipulation. Two control sutures were placed in the left ventricle, in an area devoid of coronary vessels and near the apex. These sutures were crossed and held without tension by the assistant. An incision about 7 mm. long was made in the ventricle, between the control sutures and slightly oblique to the surface of the heart. There was no bleeding. The valvulotome, having been filled with salt solution to displace the contained air, was inserted into the ventricle, and the cutting edges were placed in the mitral ring, the position being determined by feeling the end of the instrument through the invaginated wall of the left auricle. The valve was cut, and as the instrument was withdrawn from the heart, slight traction was made on the control sutures by the assistant. There was no bleeding. The incision in the ventricle was closed with two Lembert sutures of silk. The control and apex sutures were removed. The heart beat regularly; it was of good color, and was not dilated. The pericardium was closed with a continuous suture. The wound was closed in layers.

Sections of each cusp of the mitral valve were removed by the valvulotome and are shown in the inset of one of the drawings by the artist, who has made sketches from this operation (Fig. 21). There was a loud murmur extending throughout systole; the pulse rate was 200 per minute. The transverse diameter of the heart as shown by the roentgen ray was 6.9 cm. Ten hours after the operation, the heart rate was 210. There was a loud systolic murmur. The dog lay quietly in the cage, wagged his tail, and drank some water.

February 7: Heart rate, 200; respirations, rapid; no fever; when the cage was approached the dog stood up.

February 8: Heart rate, 180; loud systolic murmur; the dog walked from the cage, and ate food. It seemed that the dog might survive this marked regurgitation.

February 9: Heart rate, 190; dog lay in the cage, listless; respirations very rapid; nose cool.

February 10: The dog was found dead in the cage; its mouth and nostrils were filled with pink froth. There was blood-stained fluid in each side of the chest; 20 c.c. in the left, and 80 c.c. in the right. No pleural adhesions were found. The lungs were everywhere boggy and edematous, so that when they were cut with a knife, fluid welled out of the tissues and alveolar spaces. The pericardium was nowhere adherent to the heart; the pericardial fluid was clear and was not increased in quantity. The wound in the ventricle was well closed and healing. A part of each leaflet of the mitral valve was excised at operation, and a marked regurgitation was produced. The incised margin had healed without thrombus formation. The tricuspid valve had remained competent; there was no congestion of the abdominal organs, and no fluid was found in the abdominal cavity.

*Microscopic Examination.*—The lungs showed marked edema; the liver, spleen and kidneys were normal.

Death was attributed to mitral insufficiency, with pulmonary edema.

EXPERIMENT 18.—Dog 109, a male mongrel hound, weighing 18 kg., Feb. 11, 1924, was given one-fourth grain (0.0162 gm.) morphin; the heart rate was 80, and there was no murmur; the heart measured (roentgen ray) 7.3 cm.

*Operation.*—The usual procedure was followed. The blood pressure was taken from the femoral artery throughout the operation. The fifth rib was resected, and a segment of the mitral valve was excised and removed (Fig. 21). The blood pressure before the insertion of the valvulotome was 120 mm. of mercury; immediately after the production of the mitral insufficiency it was 94 mm. of mercury. The usual closure was made; the femoral artery was ligated and the incision sutured. The condition of the dog was good; the heart rate was 146; there was a soft, systolic murmur; according to the roentgen ray, the diameter of the heart was 7.7 cm.

February 12: Loud, systolic murmur; pulse rate, 140.

February 13: Loud murmur; pulse rate, 132; diameter of heart (roentgen ray), 8.3 cm.

February 18: One-fourth grain (0.0162 gm.) morphin given; blood pressure, systolic, 120; pulse rate, 126; diameter of the heart (roentgen ray), 8.8 cm.

February 20: Diameter of the heart (roentgen ray), 8.5 cm.

March 25: Diameter of the heart (roentgen ray), 8.8 cm.; pulse rate, 132.

May 5: Diameter of the heart (roentgen ray), 8.4 cm.; pulse rate, 144; loud, systolic murmur. The dog had mange and was losing weight. The present weight was 15.6 kg.

EXPERIMENT 19.—Dog 110, February 12, a male hound, weighing 14 kg., was operated on February 12; the heart rate was 88, and there was no murmur.

*Operation.*—The usual procedure was followed. The fifth rib was resected, and apex and control sutures placed. The incision was made in the ventricle with a scalpel, and as the valvulotome was forced through the incision the heart began to fibrillate. The valvulotome was withdrawn without the attempt being made to cut the mitral valve. Quinidin was given, but the heart continued to fibrillate.

Examination of the heart showed that the incision had entered the base of the anterior papillary muscle.

EXPERIMENT 20.—Dog 113, a female bulldog, weighing 12 kg., Feb. 21, 1924, was given one-fourth grain (0.0162 gm.) morphin; the heart rate was 84; there was no murmur; according to the roentgen ray, the transverse diameter of the heart was 7.1 cm.

*Operation.*—The usual procedure was followed. The femoral artery was cannulated for pressure determinations. The fifth rib was resected. The control sutures were placed as near the apex as possible and away from the base of the anterior papillary muscle. The incision was made and the valvulotome inserted. A large segment of the mitral valve (Fig. 21) was removed. The wound was sutured. The heart action was regular, and the color good. The femoral pressure before the sectioning of the valve was 142 mm. of mercury; after the production of the mitral insufficiency, the pressure was 124 mm. of mercury. The usual closure was made. A loud systolic murmur resulted; the heart rate was 158, and the diameter of the heart (roentgen ray) was 8 cm.

February 22: Good condition; loud murmur; pulse rate, 140.

February 26: Pulse rate, 100; good condition.

March 28: Pulse rate, 140; loud systolic murmur; blood pressure, systolic, 140, and diastolic, 70 (cuff sphygmomanometer). The dog was in a good condition.

## DISCUSSION OF EXPERIMENTAL FINDINGS

Two deaths followed the incision of the base of the anterior papillary muscle. The literature contains numerous references to the so-called vital areas of the heart, and the subject at present remains a moot question. Our experience indicates, however, that the apex of the left ventricle can be incised with safety provided the base of the anterior papillary muscle is avoided, and in operations in human cases this precaution should be taken.

One death in the series was due to hemorrhage. This was a frequent cause of death in the early experiments, in which the operative technic was being developed. Refinements in the surgical technic have shown that there need be no hemorrhage in the entire procedure, that the ventricle can be incised, the valvulotome inserted and, after the mitral valve is cut, withdrawn from the heart, and the incision sutured with absolutely no hemorrhage resulting. The control sutures should be placed with precision, just so deep, just so far apart; the traction on them should be just so much, and the incision should be accurately placed. All these are details of the first importance, and they can be appreciated only by repeated experiment on the heart.

Three deaths followed the excision of segments of the mitral valve, producing a regurgitation incompatible with life. Since the normal mitral valve is a thin and delicate structure, it becomes engaged readily between the cutting edges of the valvulotome, so that large segments may be excised and sometimes a fatal regurgitation produced (Fig. 25). A comparison cannot be made to the condition in the human heart, in which the stenosed mitral valve is rigid and stiff and in which the size of the segment removed is directly proportional to the size of the valvulotome used.

The most frequent cause of death following the operation is pulmonary edema. It occurs in those experiments in which large segments

Fig. 25.—An extensive defect in the aortic cusp of the mitral valve of Dog 92. This animal died a few hours after operation. No comparison can be drawn from the creation of such large defects in the normal valves of dogs with what might result in a human case of mitral stenosis, both because of the relatively greater size of the instrument in the case of the dog, and especially because of the difference in physical properties of the normal and stenotic cusps.

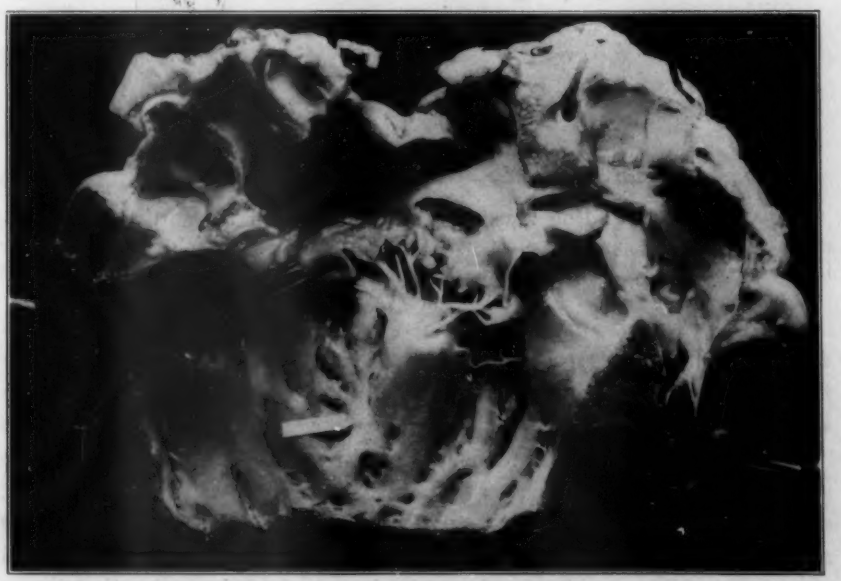

Fig. 26.—Heart of Dog 95. The animal was killed May 9, 1924, 120 days after operation for illustrative purposes; general condition excellent. The lesion is at the anterior edge of the aortic cusp and includes free valve edge and extends into auriculoventricular ring. Pointer indicates operative scar in ventricle.

of the mitral valve are removed, producing a marked regurgitation (Dogs 97, 106 and 108). In the fourteen experiments, the protocols of which are previously given, three died of pulmonary edema; this has been about the proportion in the entire series. As the heart becomes enlarged, a relative tricuspid insufficiency may develop, such as occurred in Dog 97. If the mitral regurgitation is not excessive, the dogs ordinarily do well. A few developed mange or distemper during the postoperative period and were killed; at present, from one to four months after operation, there are twelve dogs living, and, with two

Fig. 27.—Heart of Dog 96, showing two defects in the aortic cusp of the mitral valve. The smaller defect was probably due to an infolding of the valve between the cutting edges. The dog was killed sixty days after operation because of distemper.

exceptions, these are in good health. It is probably worthy of comment that, in this entire series of thirty dogs, not a single postoperative death was due to infection.

In all the illustrations in this section, in which the cardiovalvulotome was used, the defect shown is that typically created by this instrument, consisting of a removal of that part of the cusp including the free margin and extending towards the musculature at the base of the mitral ring (Fig. 26). However, in several early instances the tip of the instrument was actually forced through the cusp because it had too sharp a point, thus creating a different type of lesion (Fig. 27).

## VI. EXPERIMENTAL DATA: RESULT OF FURTHER STUDY

A. *Roentgen-Ray Studies of the Effect of These Procedures on the Size and Shape of the Cardiac Shadow.*—Roentgen-ray studies were made of the series of dogs operated on with the cardiovalvulotome and reported in Section V. Roentgenograms were taken before and immediately after the operation as well as at frequent intervals afterwards for the purpose of finding out what data might be obtained concerning the changes in the size and the shape of the cardiac shadow.[13] The roentgen-ray tube and the screen were placed at fixed distances from the heart; the former was placed at a distance of 6 feet from the screen, and the latter touched the dog (Fig. 17). By this method direct comparisons of the cardiac shadows are possible.[79]

The delicate mediastinum in the dog [80] permits easy rotation of the heart, which in turn may alter the size and the shape of the cardiac shadow. To avoid the rotation of the heart as much as possible, the dog was placed on the table in a direct anterioposterior position; even when this is carried out carefully, there is sometimes a slight margin of error in repeated roentgen-ray shadows. A number of lateral oblique exposures were taken with the idea of showing better the pulmonary artery and the left auricle, but little that was definite could be obtained, and in most of the experiments only anteroposterior exposures were made.

The cardiac shadows obtained in this series of dogs, before and immediately after the operation, show an average increase of 0.37 cm. in the transverse diameter following operation. There are variations in these measurements, from an increase of 1.1 cm. to an actual decrease of 0.2 cm. following operation. This immediate increase in the size of the heart is undoubtedly due to dilatation. It usually disappears gradually, so that the heart returns to almost, but not quite, the size that it was preceding the operation. The response of the heart to its increased work gradually results in hypertrophy, which is shown definitely by an increase in the cardiac shadow. Usually after the mitral insufficiency has been established from two to four months, the transverse diameter of the heart has increased about 1 cm.

Concerning the changes in the shape of the cardiac shadow, little that is definite can be stated. In some cases, there is a fulness in the region of the left auricle corresponding to the capacious auricle accompanying mitral regurgitation. A right or a left-sided preponderance cannot be distinguished in those cases showing hypertrophy, the shadow being a generalized enlargement. In this series of experiments, a peri-

cardial effusion was not demonstrated by the roentgen-ray shadow, and in no case was a pericarditis with effusion found at necropsy. It is the presence of a pericardial effusion forming so rapidly as to produce heart tamponade that may make the roentgen-ray determinations of the size of the cardiac shadow an important adjunct in following the postoperative course of patients. In normal dogs, however, the heart is not adherent to the pericardium, and, if there is no infection and little trauma, there is no tendency to form a pericardial effusion. It is, indeed, a striking observation at necropsy that, in almost every case, the pericardial fluid is very scant in amount and often completely absent.

Figure 28 shows serial roentgenograms of a dog's heart made before, and subsequent to, the establishment of a mitral insufficiency.

B. *Pericardial Adhesions Following Operations on the Heart.*—Following any operative procedure on the heart, the formation of pericardial adhesions is a very common occurrence. If the pericardium is simply

Fig. 28.—Serial roentgenograms of the heart of Dog 107 (protocol and Fig. 21) before and after the establishment of a mitral insufficiency; the changes in size and shape are demonstrated.

incised and sutured without trauma to the epicardium, an adhesion to the heart usually forms at the level of the suture; but if the surface of the heart is incised, an adhesion to the pericardium almost always occurs. A dense, scarified adhesion to the pericardium usually forms where the ventricle had been incised, and in only one case did the heart remain entirely free from adhesions. In about one third of the cases a few delicate adhesions to the pericardium formed elsewhere on the surface of the heart, usually at the base and at the ear of the left auricle, which had been invaginated by the gloved finger when the end of the valvulotome was felt at operation. In about one half the cases, the lung became adherent to the pericardium at the place in which the latter was sutured.

In view of the sensitivity of the epicardium and the pericardium to trauma resulting in the formation of adhesions, these structures should be handled with the greatest delicacy. All unnecessary touching should be avoided. Hot salt solution applied directly to the heart in the early

experiments, with the idea of stimulating its action, has possibly been a factor in the production of adhesions; it should only be used at operation at a temperature not above 105 F. For the same reason, the advisability of applying epinephrin directly to the surface of the heart is questionable. May it not be wiser to inject it directly into the right ventricle?

After the incision in the heart has been sutured and the oozing of blood completely stopped, the heart is elevated slightly from the pericardial cavity by the apex suture, and the blood-tinged pericardial fluid is removed by a gentle sponging with cotton pledgets moistened in a saline solution at a temperature of 105 F. The pericardial fluid is replaced by a few cubic centimeters of salt solution.

As it has already been stated, the formation of pericardial effusion does not usually occur in these experiments unless there is infection. If it were a common postoperative occurrence, the problem of handling the pericardium to avoid heart tamponade would be presented. In cases complicated by pericardial adhesions which are freed at operation, however, the tendency to produce a rapidly forming effusion, as shown in Case 3 (Section IX), exists, and the disposition of the pericardium is of great moment. If a drain is placed in the pericardial sac, it would undoubtedly be followed by pericardial adhesions so that, as it became sealed off, the drain would no longer drain. Larrey,[15] in 1829, observed the presence of adhesions surrounding the drain in several cases in which he performed pericardiotomy for the removal of foreign bodies; but these cases were undoubtedly complicated by the presence of infection.

The pericardium shows a great tendency to form adhesions to the heart and lungs. Almost always, the pericardium becomes adherent at the site of the incision of the heart, and sometimes the adhesion is very firm. Less often, the lung adheres to the pericardium at the line of suture.

C. *The Healing of Wounds in the Pericardium, Myocardium and Endocardium.*—The primary healing of wounds in the heart was first studied by Bloch,[19] Del Vecchio,[20] Salomoni,[21] Bode [22] and Elsberg.[30] For many centuries the heart was considered the seat of the soul, and to touch it meant instantaneous death. Surgical thought, accordingly, did not go beyond that point, and the idea that there was a possibility of a wound of the heart healing was not conceived until the nineteenth century.

Towards the end of the century, however, and especially after the demonstration of the feasibility of suture in the human heart, the healing of wounds was observed closely and the histologic picture studied under the microscope. It was found, as one would naturally expect,

that wounds of the heart could heal by first intention, and that they were capable of producing a firm and sufficient union, with a histologic reaction comparable to that of muscle wounds elsewhere. A discussion of this subject is unnecessary here. Suffice it to say that a strong union of the muscle occurs and that, histologically, there are present fibroblasts, mononuclear wandering cells, and connective tissue cells, the whole picture becoming resolved later into a firm scar. It is not infrequent to find the scar calcified; this finding was noted also by Cushing and Branch. A few references are found in the literature concerning the suture material preferable for use in the closure of a cardiac wound, but there can be little question as to the superiority of silk over catgut for this purpose.

The endocardium also heals by first intention, producing a scar covered with smooth, glistening endocardium. There is no thrombus formation; not a single thrombus was found in the many wounds that we examined, either in those that were in the process of healing or in those wounds that were scarified and contracted.

The incised margins of the valve also heal without thrombus formation. The healed margin of the valve is often thickened and rumpled, sometimes attaining a thickness of 1.5 mm. This thickness, however, is due not so much to a cellular reaction as to a turning-in of the incised margin so that the edge represents a doubling of the valve. It is this tendency that causes an operative lesion of the normal valve to actually increase in size as healing progresses. Histologically, fibroblasts, round cells and connective tissue cells are present, sometimes in sufficient numbers to cause a little thickening, and the healed margin is covered with endothelium.

D. *The Reaction of the Heart to Trauma.*—The immediate reaction of the heart to the operation of partial mitral valvulectomy is shown in Figure 29. The tracing indicates pressure changes in the femoral artery. A number of wide excursions in the arterial pressure are seen when the ventricle is incised, when the distal end of the valvulotome is felt by invaginating the auricle and when the incision is sutured. These wide swings in pressure are probably due to extrasystoles, each of which is followed by a forceful contraction of the ventricle. The curve indicates also the remarkable ability of the heart to tolerate incision and subsequent suture of the wound, both the blood pressure and the rhythm being well maintained in spite of the presence of an added mitral regurgitation. The tolerance of the human heart to trauma has been demonstrated repeatedly at operation in cases in which extensive wounds have been sutured. In the remarkable case recorded by Travers,[81] a trifid wound (Fig. 30) in the right ventricle 2¼ inches long extending into the interventricular septum was sutured, in the process of which

Fig. 29.—The pressure changes (mercury manometer) in the femoral artery of a dog during the operative procedure consisting of incision of the left ventricle, insertion of the valvulotome, excision of a segment from the mitral valve, and suture of the wound of the heart. The pressure is well maintained throughout the procedure. The pressure after establishing mitral insufficiency is somewhat lower than the normal pressure, and the pulse pressure is somewhat decreased. The time is shown in seconds.

Fig. 30.—Traver's remarkable case of a trifid wound of the right ventricle, two and one quarter inches long, sutured at operation. In the process of suture the tips of three fingers plugged the rent to control hemorrhage. The heart stood the operative trauma, but death occurred eleven days after the operation, due to a clot in the pericardial cavity (from the *Lancet*).

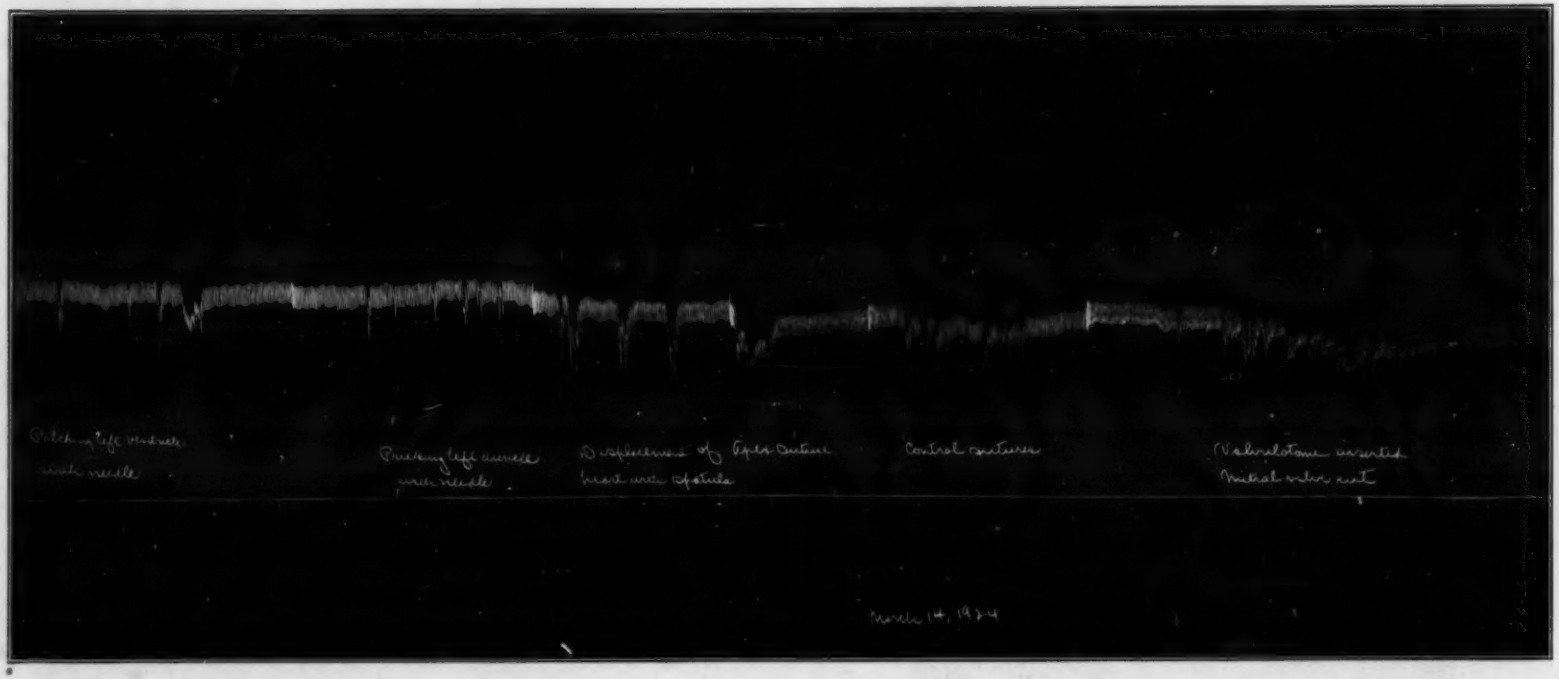


Fig. 31.—Pressure in the femoral artery of a dog with a mercury manometer showing the effect of pricking the left ventricle and auricle with a needle, displacement of the heart from the pericardial cavity, the placing of sutures in the myocardium, and the insertion of the cardio-valvulotome. The irregularities recorded are probably due to extrasystoles. There is no evidence that these manipulations seriously embarrass the heart.

the wound was plugged by the surgeon's three fingers to control hemorrhage. The patient lived eleven days, death being due to a clot of blood forming in the pericardium.

Mechanical stimuli such as the pricking of the ventricle or the auricle with a needle, or the displacement of the heart from the pericardial sac with a spatula (Fig. 31), are usually followed by abnormal contractions, probably extrasystoles. This response of the heart varies considerably in animals. In one experiment, Dog 105, the heart was most sensitive, simple displacement from the pericardial cavity with a spatula producing a tachycardia which lasted several minutes.

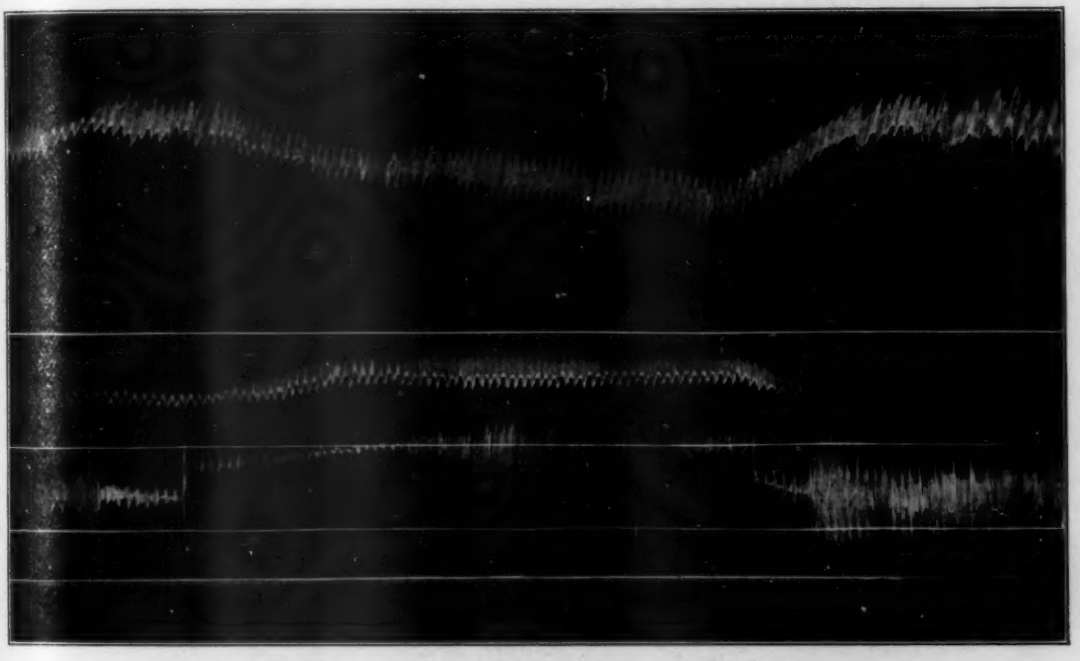

Fig. 32.—The effect of heart tamponade. The upper curve shows the arterial pressure, femoral; the middle curve shows the venous pressure, innominate; and the lower curve shows the intrapericardial pressure. The intrapericardial pressure is increased 8 cm. of water and causes a marked fall in arterial pressure and a rise in venous pressure. The time is shown in seconds.

The ability of the heart to withstand section of the mitral valve depends largely on the degree of the regurgitation. If the regurgitation is marked, the heart dilates, becomes cyanotic and often irregular; if ventricular fibrillation occurs, death follows. The heart responds to the regurgitation by an increase in rate, in some cases two hundred beats per minute, which, in the course of a few months, produces hypertrophy of the heart. Aortic regurgitation may be well tolerated also, as has been shown in the cases discussed in a previous section.

The tolerance of the heart to incision depends, as it has already been stated, on the part of the heart incised. One would judge from the work of earlier observers that the region of the auriculoventricular ring contains structures that may not be incised; our experience indicates that the base of the anterior papillary muscle should be avoided.

It is obvious that the coronary arteries and their principal branches should be avoided, not only on account of hemorrhage in the operative field but also because of their importance in the maintenance of the normal rhythm of the heart (Porter [82]). Localized infarctions might occur if important trunks are ligated.

Traction and displacement of the heart usually give rise to extrasystoles and a slight fall in blood pressure. In some cases, however, traction has little or no effect on the rhythm or the blood pressure. In an experiment performed on a dog, a weight of 200 gm. exerted traction on the heart through an apex suture, and at the end of ten minutes no appreciable change in the blood pressure occurred.

The importance of heart tamponage was early appreciated; Morgagni,[17] in 1761, for the first time, and Franck,[83] in 1877, described its dangers. The term "Herz Tamponade" was coined by Rose [16] in 1884, and since that time Cohnheim,[57] Lewis,[84] Kuno,[85] and others have studied its physiology. Its rôle is of great importance in cases of pericardial effusion, and after the pericardium is opened at operation the formation of an increased pericardial tension merits close study. In the experimental work, pericardial effusions and, therefore, heart tamponade did not occur except in one case in which radium emanations had been placed in the mitral ring for the purpose of producing stenosis. In this case, the wall of one of the coronary veins had become ulcerated, so that bleeding into the pericardial sac occurred, producing death. We have, however, conducted experiments that emphasized to us the danger that such a condition might cause to arise after operation in a human case. Figure 32 illustrates this point.

## VII. A DISCUSSION OF THE VARIOUS SURGICAL METHODS AND THEIR APPLICABILITY TO THE TREATMENT OF MITRAL STENOSIS IN MAN

We have already discussed the rationale supporting the proposal that certain cases of mitral stenosis might be benefited by a surgical procedure. It is now our task to select from those methods either reviewed in the historical section or elaborated through our own experimentation the one that seems to embody both the greatest accuracy in the performance of the desired task and the greatest safety to the patient in its execution. It is apparent that all the procedures described can be divided into two main groups: Group I, in which the valves are cut under direct vision with the heart opened and the circulation shut off by compression of the great vessels at the base of the heart; and Group II, in which the valve is sectioned with the circulation intact. Obviously, the technical procedure in Group II is the more difficult, as it presupposes that methods can be evolved for handling a writhing and pulsating organ and for insuring against the great danger of hemorrhage.

Both in our own experience with animals and according to the experimental work of others, it was evident that the entire circulation could not be shut off longer than three minutes if full recovery was desired. The reasons for this are based on the limited resistance of certain nerve cells to a continued anoxemia. Although vital nerve centers, such as the medulla, will stand a far longer period of anoxemia, certain of the cortical cells degenerate rapidly, and thus, although the animal may recover after three to four minutes, it remains partially if not totally decerebrate. In human cases, Sauerbruch's experience in clamping off the venae cavae during the closure of wounds of the heart, especially if compression is applied intermittently, has shown that probably there is greater tolerance to this procedure in man than in the lower animals. Even so, the time that could be devoted to an intracardiac procedure would be very short. This is most important, because, if there is one quality vital to the successful performance of such procedures, it is equanimity at the moment when the valve is to be attacked. Any sense of impending disaster such as would occur when the procedure must be performed with great rapidity, would rob the operator of this quality, "aequam servare mentem." Moreover, in cases of mitral stenosis, there is always some muscle damage; it is only reasonable to suppose, therefore, that such hearts would be less likely to withstand the period of complete occlusion of the circulation than normal hearts. Because of these objections, we feel justified in dismissing from any further consideration those procedures requiring occlusion of the circulation, in spite of the obvious benefit to be derived from carrying out the sectioning of the valve under direct vision.

In Group II, we must consider both the medium of approach into the heart and the instrument to be used. The question of danger zones has been discussed, and there is every indication that the passage of instruments either through the apical portion of the left ventricle or through the left auricular appendix bears no inherent risk. Neither our own experience nor the experience of previous investigators agrees with the contention of Allen and Graham that the ventricular approach offers any special risk. Indeed, the fact that this portion of the heart may be excised successfully, with recovery resulting, is the best indication of its tolerance to wounds. The equal safety of the approach through the auricular appendix, used by MacCallum and Allen and Graham, has also been demonstrated in this laboratory.

In choosing between the left ventricle and the left auricular appendix as the avenue of approach, we feel that the weight of available evidence at this time is in favor of the former. Or rather, we feel that there are several reasons against the use of the appendix approach. This is based on the fact that in mitral stenosis there is present a damming back of the blood into the auricle. This means a slowing of the current, especially emphasized in such eddies as the appendix. Eventually, there occurs, at least in cases of long standing and marked stenosis, dilatation of the auricle. In fact, unless this dilatation is present, one would question the diagnosis of mitral stenosis. These two factors, stasis of the blood stream and dilatation of the auricle result in two dangers: first, that either an old thrombus may be found occluding the appendix, such as is commonly seen at necropsy, or a fresh thrombus, which would be dislodged by the insertion of an instrument; second, the great risk of a tear in the greatly distended and thinned-out auricle. Moreover, even if successful, one would be confronted with the danger of subsequent thrombosis and embolism resulting from injury to the intima. Certainly, this would be more likely with the slower flow of blood in the auricle, than in the ventricle. We feel that any one who has viewed the thinned-out, dilated bag, for it is thus that the left auricle appears in the living patient, would refrain from an attempt to insert an instrument into it. To be sure, the ventricle has the disadvantage of being more active in motion; but its thick walls permit safer handling, and it is more easily repaired. Furthermore, the ventricle is more accessible, even with a transpleural approach. This latter procedure, which will be discussed later, and which involves a further tax on the capacity of the lungs to ventilate themselves, must be deprecated. According to such reasoning, the left ventricle seems to offer the safest region for the insertion of the instrument to be used.

In discussing the actual instrument with which the final step is to be taken, we must at once choose between the use of one that affords a certain amount of vision by means of a tube with a lens and

light at its extremity, and one without that advantage. Naturally, all surgeons will deprecate a method in which a vital step is taken without the aid of the eye. However, in establishing new procedures, we must consider the patient as well as the point of view of the operator.

The cardioscope of Allen and Graham has been described earlier in this communication, and our own attempts with a similar instrument are discussed in Section IV. It is not of great importance that we do not fully agree with these investigators who believe that such an instrument gives sufficient clarity of vision, which is an aid in both an appreciation of the pathologic state and in accurate orientation. Any one familiar with any endoscopic method, even in a satisfactory, clear medium such as is present in cystoscopy, thoracoscopy and bronchoscopy, will agree that at least some time must always be consumed in orientation. How much more time will be necessary when the medium in which the instrument is placed is opaque and when vision is only afforded with a minimum of the medium between the lens and the object to be examined, such as is possible in this method only by contact of lens and endocardium, can well be imagined. Moreover, this presupposes that one can recognize the structures thus brought into contact. Is such familiarity with the inside of diseased hearts possible, and where is one to obtain the practice that will enable one to develop judgment as to where the tip of the instrument may lie in the most efficient and reliable position? Without a great amount of practice, one can never hope to develop sufficient familiarity to orientate one as quickly as it is possible for Chevalier Jackson to do in his bronchoscopic work; yet no one would deny that, in the field of surgery under discussion, rapid and reliable orientation would be even more desirable than in the other field just mentioned. Surely, a great deal of time will be consumed by the necessary searching about. Furthermore, this method brings up a purely technical matter of moment. In none of the other endoscopic methods is the question of perfect asepsis a requisite of success. In this field, however, it may be a question not only of success, but of life. Is it safe to put even a carefully masked face close to an open pericardium, to say nothing of the possibility of contaminating the hands or the danger from the eye-piece against which the orbital tissues must be placed if the maximum amount of information is to be obtained by this search? In view of the investigations cited above and of the experiences in Cases 2 and 3, which are to follow, are we to hope that a delicate knife, such as is carried in this instrument, even if it can be accurately placed against the stenotic valve, can sufficiently enlarge the opening?

Having thus rejected the approach by way of the auricular appendix as well as the use of the endoscope, we must now consider what sort of an instrument offers most promise in an attack on the stenotic orifice through the left ventricle. We have reviewed the physiologic instru-

ments consisting of small knives or hooks on long handles with which normal valves in animals are easily injured. We have described the success of surgeons in perfecting methods by which these instruments can be inserted directly into the heart by way of a thoracic approach. We have reported our own experience with such instruments, and it was this experience that led us to use this method in the first three cases subsequently reported in this communication. The objections to these small knives are fully cited and discussed in Section V, and were seen to be corroborated by the examinations of the hearts in Cases 2 and 3 after operation. It was our failure to greatly reduce the stenosis in the latter cases that led to the development of the cardiovalvulotome already described. In Section V, the efficacy of this instrument is shown both in attempts made on typically stenosed hearts removed at necropsy and in animal experiments. These experiments bring further evidence to bear on the safety of the apical portion of the left ventricle as an avenue of approach.

These considerations are sufficiently valid to lead us to state that, in the present status of our knowledge, it seems wisest to use the apical portion of the left ventricle as the safest locus for the insertion of an instrument into the left side of the heart in cases of mitral stenosis. We feel that the cardiovalvulotome, which we have described, is better suited to the task of enlarging a mitral stenotic orifice than the other methods heretofore elaborated. The technical performance of operations in man for the reduction of the stenosis in cases of chronic mitral valvular diease, in which these considerations are accepted, is described in Section VIII.

## VIII. THE OPERATIVE PROCEDURE IN HUMAN CASES

The things relating to surgery are: the patient; the operator; the assistants; the instruments; the light, where and how; how many things, and how; where the body, and the instruments; the time; the manner; the place.—*Hippocrates.*

The determination to attempt a reduction in the stenosis in chronic, mitral valvular disease necessitated the choice of what might constitute the best method for exposing the heart, preparatory to the actual attack on the valve itself. In making a choice of one of the many methods of exposure already described or in elaborating a new technic, it was necessary to take into consideration the fact that the heart must be fully exposed, so that the left ventricle is easily brought into view with a minimum of trauma, handling and dislocation, in order that in the event of a technical accident the operator may be afforded every opportunity to control hemorrhage. Nothing short of complete exposure of the entire organ will suffice. The only other consideration of prime importance in this relation is the selection of an exposure so designed as to interfere as little as possible with the ventilating capacity of the lungs;[78] these organs are already engorged with more or less stagnant blood, and any further factor contributing to the reduction of their capacity may immediately result in disaster.[86] It was because of this latter consideration that we discarded from the beginning all methods that necessitated a transpleural approach.

The objection to the transpleural approach limits the procedures to the midline, because the incision must be so placed as to open the pericardium where it is not covered by the pleurae. One may divide the operations already devised with the same object in mind into two groups: first, the parasternal operation, in which an incision, either oblique or perpendicular, is made at the left lower sternal edge and either one or several contiguous costal cartilages excised, or in which an osteoplastic flap is turned outwards (Delorme,[87] Ollier,[88] and Kocher;[89] second, the midsternal operations, in which the sternum is split in its midline (Milton,[90] Rehn,[26] Duval and Barasty[91]). The operations of the parasternal type were devised chiefly for the drainage of the pericardial cavity, though they have been adapted from time to time for the extraction of foreign bodies and for the suture of wounds of the heart. All the procedures in this group have two important limitations: first, they only expose a small portion of the heart to view, and they do not allow room enough for dislocation of the heart and examination of its posterior and more remote aspects; second, they always introduce the possibility of opening the pleura, especially if a trap-door flap is utilized or wide traction exerted. Indeed, the small amount of space provided by these incisions is of no more practical use in a procedure such as is

contemplated for the relief of mitral stenosis than the trephine hole of Riolan,[92] 1648, or the burr opening of Terrier and Reymond,[93] 1898.

The midsternal route is the only method that fulfills the requirements in operations performed on the heart valves. Median sternotomy as originally proposed by Milton,[90] of Cairo, consisted in longitudinal division of the sternum from the sternal notch through the ensiform. This incision, although it gives a better exposure than the parasternal methods, fails to open the thorax widely, even with great traction, because of the constricting action of the clavicles and the diaphragm. Rehn modified this method by cutting across the sternum below the clavicles and splitting it from that point down in the midline. Such an exposure is only fairly satisfactory, because the apical portion of the heart is distant and difficult of delivery. The final step in the perfecting of an exposure that reveals the entire heart and that permits ease in the handling of the organ with the minimum of trauma and dislocation was added by Duval and Barasty.[91] They suggested division of the diaphragm and of the linea alba at the inferior aspect of the wound. This latter step, after midline sectioning of the sternum and its horizontal division at the level of the second or third interspace, makes it possible for the wound to open as widely at the top and the bottom as it does in the center where traction is exerted. We have used such a procedure in all four of our cases, the first of which has already been reported. It has proved entirely satisfactory. The cases reported by Duval and Barnsby,[94] Barbier and Goujon,[95] by Rouvillois [96] and by Tuffier,[27] in which foreign bodies were extracted, provide evidence both of the amplitude of the exposure and of the ability of the patients to withstand the ordeal. The incision appears formidable in description but we would recall that, though large, it is placed in the midline where there are no great vessels or nerves to be injured, and where the pleurae need not be opened. In our own experience, we feel it has not added any special risk, nor has it taxed the vitality of our patients. We have added certain technical changes; thus, we have, in all cases, cut the sternum across at the level of the second interspace (Duval and Barnsby used the fourth, and Rouvillois the third space), and we now use a motor driven circular saw with which to divide the sternum (in Cases 1 and 2 we used a Hudson osteotome, and in Case 3 burrs and Gigli saws).

The details of the procedure are as follows: The patients are given a small preliminary dose of morphin and atropin. Ether anesthesia [97] is induced by the open mask method; when it is complete, a catheter is led into the nasopharynx through a nostril, and anesthesia is continued by the carrying in of ether vapor through this tube by means of the Connell ether machine. This method of producing anesthesia permits distending of the lungs, if the pleurae should be accidentally injured, merely by

shutting off the outlet by way of the mouth and the other nostril. The patient is placed in a semirecumbent position with the head and thorax elevated to an angle of 30 degrees. The skin is prepared by shaving and scrubbing with sponges wet with 70 per cent. alcohol and 1 : 1,000 mercuric chlorid. The line of the incision is then scratched on the skin with a scalpel (Fig. 33) from the sternal notch to a point midway between the ensiform and the umbilicus. Cross scratches are made at several points to facilitate accurate skin approximation later in such a long incision. Toweling is applied up to the line of incision and the draping

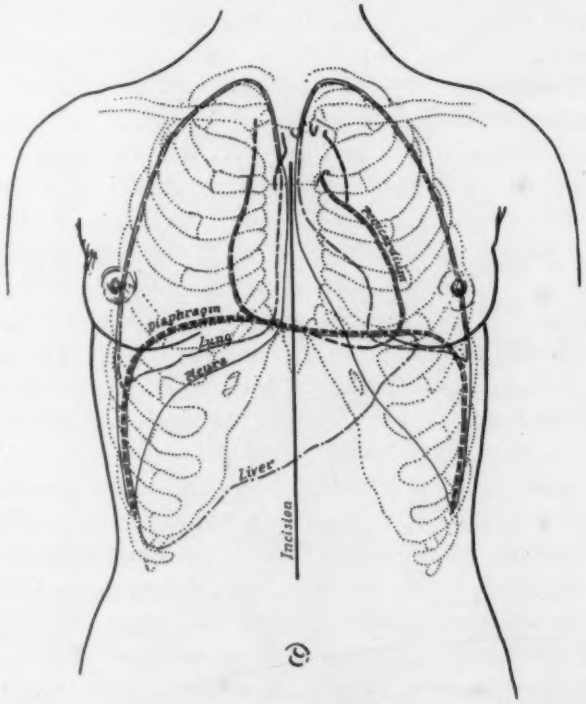


Fig. 33.—Line of incision.

completed. The incision is then carried down to the bone at once, and inferiorly through the linea alba. The ensiform is freed of its attachments and removed (it is easier to remove it than divide it, and the closure also is facilitated). The second and third fingers of the right hand are then thrust below the sternum at its inferior edge, and the tissues there are pushed away from the bone. It will be found that inserting these two fingers together and then spreading them is very efficacious. As one gets above the lower limits of the sternum, these attachments are much less dense, and progress is very rapid. Finally, a flat spatula, such as is used in the gasserian ganglion operation, or even a narrow

ribbon retractor, is inserted in place of the fingers beneath the sternum, care being taken to keep all loose and adventitious tissue below this instrument. This spatula acts as a protection against accidental, sudden perforation of the sternum. It should be held by an assistant.

The sternum is then split from below upwards with a motor driven, circular saw. We think it important to use a saw as it gives excellent reapproximation for the closure, and this results in a diminution of pain in the twenty-four hours immediately following the operation. If a bone cutting instrument is used, an irregular edge results, which, because of the rubbing together produced during respiration may result in considerable pain (Case 1). As the sternum is split, assistants on each side lift upward and outward by pulling on small retractors. At the level of the second interspace, the sternum is cut across with the saw, care being taken that the mammary arteries are not injured. With the assistants still exerting a pull upward and outward, the attempt is then made to wipe the parietal pleurae away from the chest wall for a short distance on both sides. This is most safely done by means of large pledgets of wet cotton. It is now possible to see the line where the pleurae overlap the pericardium. A stout, self-retaining retractor of the Tuffier or Sauerbruch type is now inserted, and supplants the separate retraction on the split halves of the sternum. The two halves will be seen to bend upward a little, because of the elasticity in the costal cartilages. The beating of the heart can now be seen through the pericardium, unless there is much fat or fibrosis of the sac itself.

The pericardium should now be opened with a knife between forceps, to begin with, and later with scissors, throughout its entire anterior aspect. A few silk sutures placed at cut edges and weighed by hemostats will suffice to keep it widely opened. Next, the peritoneum over the liver is opened, the cut being carried up to the diaphragm, down to which the opening in the anterior pericardium has been already carried. The abdominal cavity is closed to view by a warm, wet pack. Finally, the diaphragm and inferior pericardium are slit together inward for about 2½ inches (6.35 cm.). With this last maneuver, the wound opens up greatly and the self-retaining retractor should be sprung widely open. One should now have in view the greater portion of the right ventricle, a portion of the right auricle, the tip of the left auricular appendix and the anterior part of the left ventricle (Fig. 34). The apex is still hidden to view, but it can easily be delivered. We have found that some handling of the heart must be done; we even believe it a salutary proceeding, because our experimental work seemed to indicate that a heart about to be subjected to considerable trauma will withstand that trauma better if it has been somewhat traumatized previously. It is as if gentle trauma had a partially immunizing effect. Therefore, we have always

rotated the apex upward and to the right, delivering it well into view and placing a silk mattress suture, untied, close to the apex, to be used as a handle for changing the position of the organ later.

At this stage, it is well to observe carefully the heart action, and to wait perhaps a minute. A little warm saline may be dropped on the heart from time to time. One is now prepared to complete the procedure. The operator should deliver the heart to a position satisfactory

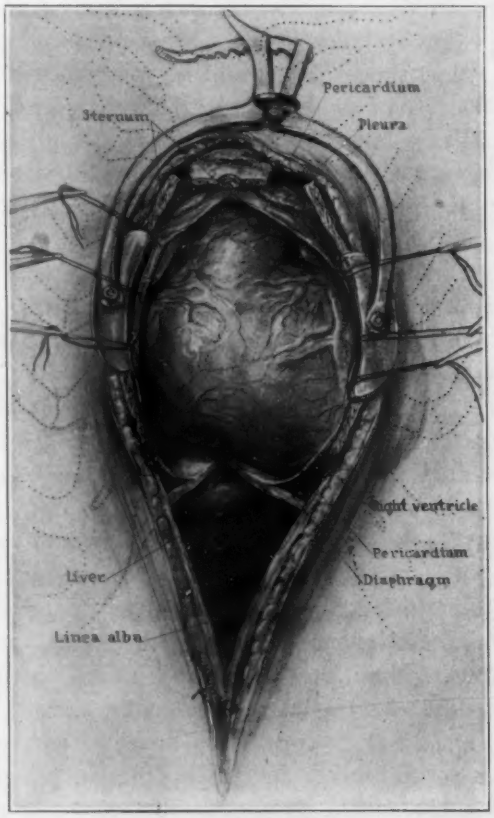


Fig. 34.—The final stage in the exposure of the heart. A warm pack should be inserted over the liver to wall off the abdominal cavity. When the sternum is divided, a little benefit is gained by keeping the incision somewhat to the left of the midline. Further benefit is obtained by keeping the division of the diaphragm also to the left. This drawing was made from Case 1, in which burrs and Gigli saws were used to divide the sternum.

and comfortable to him by pulling on the apex stitch. In order to orientate one's self, it is necessary to see a large portion of the wall of the left ventricle; this view can only be obtained by pulling the apex upward and to the right, and by slightly rotating the whole organ to

the right. This manipulation is well borne and should cause no change in the action of the heart. The operator then gives the apex stitch to an asistant to hold. This is a delicate performance for the reason that, with each beat of the heart, the assistant's hand must give way a little and move in rhythm or else the suture will tear out. Two control sutures of silk [98] are then placed in the apical portion of the left ventricle about 1 cm. apart; these threads are crossed and the ends given to the first assistant. The point of a scalpel is inserted into the heart muscle (Fig. 18), between these sutures, and then the cardiovalvulotome is pushed into the cavity of the left ventricle in the closed position (Fig. 19). This instrument is held in the operator's right hand. With his left index finger he may invaginate the auricle and feel the mitral orifice, or at least orientate himself as to its location; then, by directing the tip of the instrument towards his left index finger, there should be little difficulty in inserting the tip of the instrument into the stenosed ring.

There are, unfortunately, many dangers and difficulties at this stage. If the stenosis is marked and of long standing, the orifice will be sucked well down into the cavity of the ventricle (fish-mouth type); then, too, the auricle may be tremendously dilated and have very thin walls. If such is the case, it would be foolhardy to push down too hard with the left index finger. However, the sense of feeling is of great value, and one is easily assured when the tip of the instrument is engaged. At this moment, the spring in the handle is released and the jaws of the instrument open. Then when the instrument is inserted further, there is a sudden jar as the distal piece goes through the orifice, and the proximal blade is caught from following it by the toughened stenotic ring. This sensation is definite and unmistakable. At this point, one should exert light pressure toward the aortic cusp of the mitral valve as this takes the instrument away from the lateral wall of the heart. The blades of the instrument should now be closed. With the instrument kept tightly closed, it is slowly withdrawn. As the tip emerges, the control sutures are tightened. There should be no bleeding. Two or three Lembert sutures can then be carefully placed between the control sutures, and the difficulties of the procedure are over (Fig. 20). The control and apex suture are removed, and the heart is allowed to assume its natural position in the pericardial sac.

If the cardiac action should show signs of failure, with fibrillation, it has been proved efficacious to drop on it some warm normal salt solution. If this should not suffice to establish a better contraction, 1 c.c. of a 1:1,000 solution of epinephrin added to a few cubic centimeters of the salt solution should be used; if total stoppage is threatened 1 c.c. of a 1:1,000 solution of epinephrin can be dropped directly on the

heart or even injected into the cavity of the right ventricle. As a last resort, manual massage should be employed, care being taken to do this gently and to relax the grip entirely between each contraction of the hand in order to allow free flow in the coronary vessels and to complete the diastolic filling of the chambers of the heart. It is our impression that such a manipulation will always succeed, unless a vital center has been actually destroyed (Case 2).

Closure may now be begun, but first one must be absolutely certain that there is not even the slightest oozing of blood between the sutures at the point where the opening in the heart was closed. One of the gravest dangers will always be a postoperative hemopericardium with its resultant heart tamponade (Case 3). Moreover, it seems as if the presence of any blood in the pericardial cavity encourages the formation of adhesions. The slit in the diaphragm and inferior pericardium is first closed with a continuous silk suture; next, the peritoneum is closed and then the linea alba. This gives an opportunity to observe the heart action for some time. Now the anterior pericardium is closed with a running suture of fine silk. The fact that an aseptic traumatic pericarditis with effusion is probably certain to occur, together with and the dangers of hemopericardium and heart tamponade have led us into a thorough discussion of the question of complete closure of the pericardium or its drainage. We are in agreement with the most modern conception regarding this, and feel that complete closure is probably the wisest step to take. This question, however, is by no means settled in spite of the recognized fact that, with drainage, we are more certain to have cardiopericardial adhesions. The final closure is simple. The two halves of the sternum are allowed to settle back into apposition and are maintained there by two silver wire ligatures. The subcutaneous tissues and the skin are closed with interrupted silk sutures, and a silver foil dressing and a snugly fitting, but not too tight, chest swathe is applied.

For purposes of record and information we have kept charts during the operations on which pulse and respiratory rates and blood pressure levels, both systolic and diastolic, have been frequently indicated. In two cases, we have run frequent electrocardiograph observations. From the latter, little has been learned; but the picture presented by the former is of the greatest value (charts of Cases 1, 2, 3 and 4).

The procedure appears formidable and has been quaintly called the necropsy incision. However, every one who has used this technic agrees on the absence of shock and the ease with which the performance is tolerated. Moreover, it fulfills that primary requisite of good surgery, namely, ample exposure both for the necessary manipulation and for

the control of any accidents which may occur. Furthermore, it is not a very time-consuming procedure, provided the bony structures are divided with a motor driven saw.

We have found it of great value both in the cases of animals and in our human cases to have before us serial roentgenograms, in order to watch the changes in the size and shape of the cardiac shadow. Dr. Burgess Gordon, resident physician at the Peter Bent Brigham Hospital, has designed a way-bed,[98a] in which the patient can be elevated or lowered by means of a crank and cogs, and in which he has replaced the mattress at the back of the patient with a piece of canvass stretched taut, as in a Bradford frame. Our patients have all been kept in such a bed and, without any disturbance to them, frequent roentgenograms have been made by the use of a portable roentgen-ray machine, with the patient always at the same angle, and the tube at the same distance (about 1 mm.) from the target. By this method, one can make roentgen-ray studies daily, and by comparison with preoperative studies made in the same fashion much information can be obtained. This information thus obtained, plus pulse and blood pressure readings, furnishes the most reliable evidence that can be secured in the first few postoperative days.

The one other consideration that should be discussed at this point is that of medication; this concerns both the preoperative and postoperative use of drugs. As a rule, the action of a diseased heart is improved by digitalis therapy.[99] As a principle, it is a mistake in medicine as in war to utilize all reserves with the original attack. However, we must remember that in the case of digitalis, which is probably the most efficacious drug in cardiac therapy, a considerable amount of time is consumed before its full effect is noticed, if it is given by mouth, which is the customary method employed. But oral therapy may be impossible after operation, because of gastric intolerance and vomiting following the use of the anesthetic. Therefore, we believe cases of mitral stenosis, before being submitted to this procedure, should be thoroughly digitalized. As a postoperative drug producing maximum effect in improving the circulation, caffein easily has proved the most reliable. We recommend its use frequently if necessary. Sometimes coffee enemas have been as satisfactory as intramuscular injections, unless the occasion is imperative. The use of epinephrin should be discouraged, since the temporary elevation of blood pressure is usually, if not always, followed by a return to an even lower level of pressure within an hour.

## IX. FOUR CASES OF MITRAL STENOSIS TREATED BY SURGICAL METHODS

These four cases constitute the first attempts to enlarge the stenosed mitral orifice in human patients. A preliminary report of Case 1 appeared in the *Boston Medical and Surgical Journal* of June 28, 1923,[100] one month after the operation. We feel that, in view of the brief report given at that time and because the patient is still living, it will be of interest to report it again in full together with the three subsequent cases.

These cases are not, however, the first attempts to operate on man for valvular disease. In 1912, Tuffier [27] operated on a young man with a marked aortic stenosis. He states that he would have liked to attempt division of the valve, but that he thought that the experimental data justifying this was insufficient. Instead, he attempted to dilate the orifice by the insertion of his little finger. He reported that there was temporary improvement, and that in 1920 the man was still living. Tuffier also reported [101] that Doyen attempted to operate on a patient considered to have mitral stenosis, but that the operation disclosed an interventricular communication. The patient did not recover.

In presenting this work as a whole, it has been impossible, for obvious reasons, to adhere throughout to a strict chronologic order. In fact, any attempt to do so would have only confused the reader. Hence, the present system of grouping all experimental and clinical data separately was adopted. It seems best, however, to call to your attention at this point that the experimental and clinical work, except for the early investigations recorded in Section IV, were going on simultaneously. Thus, in the first three cases, the knife method was used in the dividing of the valves. However, after the postmortem examination in Case 2, we became convinced that this method would not prove satisfactory, and we began at once the development of the cardiovalvulotome described in Section V. The principle of excising a fragment of the valve had been in our minds for at least a year. The new and powerful valvulotome was actually sterilized and on the table in Case 3, but as this case was complicated by an adherent pericardium, we felt it unwise to use it when the opportunity to do so arrived. An hour's struggle with the pericardial adhesions had unfortunately dissipated much of our courage. The new instrument, however, was used in the last case and proved effective. With each clinical attempt, new questions arose which could only be answered by fresh laboratory investigation. The experience in such work enabled us to approach each new patient with both better instruments and an improved procedure. Carrel once stated that many of the questions put to him regarding the possibilities of the surgical treatment of valvular disease could only be answered by experience

obtained in human cases. It is our hope that we have obtained as much benefit as our capacities enable us to obtain from the following attempts in human cases:

## REPORT OF CASES

CASE 1.—*History.*—E. L., a school girl, aged 11, was admitted to the Peter Bent Brigham Hospital, May 17, 1923, with dyspnea, orthopnea, palpitation and hemoptysis. The patient's father was living, but he had cirrhosis of the liver. The mother, two brothers and two sisters were living and well. The great-aunt on the father's side died of diabetes. There was no known family history of cancer, gout, tuberculosis, hemophilia, arthritis, or diseases of the circulatory, respiratory, digestive, renal, nervous or muscular systems, except that noted above. The patient was born and had always lived in Boston. She had had chickenpox, measles, mumps and whooping cough as a child, and growing pains in the arms and legs several years before, although the mother denied that the child had ever had rheumatic fever or swollen joints. There had been no chorea, but there had been frequent sore throats previous to tonsillectomy performed six years before. Influenza had occurred five years before. There was no history of smallpox, diphtheria, chorea, rheumatism, pleurisy, pneumonia, malaria, scarlet fever or typhoid fever, and no known association with tuberculosis. There had been no injuries, and tonsillectomy and adenoidectomy had been performed six years before at the New England Hospital. The present illness dates back about three years, when the patient began to have shortness of breath on such exertion as running and climbing stairs, and was obliged to rest during play. About this time, while the patient was dancing at a festival, she had become so short of breath that she had to stop. She remained in bed three days, but the breathlessness returned on exertion, and the patient had a cough. About this time, she complained of eye ache, pain in her arms, occasional headache and pain in both ears, and a discharge from abscesses in one ear. No ear trouble had occurred since. The patient visited the Children's Hospital twice and then spent three weeks in the hospital for the purpose of resting, which seemed to improve her. The winter of 1921-1922 was spent with her grandmother in Rhode Island. While she was there, she had occasional coughing spells; these would come on suddenly, and while they lasted, she was markedly short of breath and could not lie down in bed. The patient could not play like other children; she had to rest frequently, and there was marked dyspnea on such exertion as climbing stairs. After that she progressed fairly well for eight months, up to October, 1922, when she began to have palpitation, increasing breathlessness, and, on one occasion, a definite hemoptysis, raising about a tablespoonful of bright blood. She was sent to the Boston Dispensary where she stayed one month; nine months before her admission to the hospital, she was transferred to the House of the Good Samaritan, where she remained six months. A physical examination made there showed marked bulging of the precordia, diffuse and forceful apex impulse, with a diastolic thrill. The limits of percussion dulness were: 2.5 cm. to the right of the midsternal line, and 9.5 cm. to the left of the midsternal line. There was a crescendo, rumbling murmur leading up to the sharp first sound, and a faint systolic murmur. The liver and spleen were not felt. There was no ascites or edema. The diagnosis of mitral stenosis was made. Six months before her admission, without any warning the patient coughed up a considerable quantity of bright blood. There was no pain in the chest; hemoptysis was repeated several times that day. The lung findings were nega-

tive. During her stay in that hospital, the patient coughed up blood on several occasions, and about four months before admission she had a hemoptysis in which she spit up about 300 c.c. of bright blood. May 17, the patient was transferred to the surgical service of this hospital after consultation with Dr. Maurice Fremont-Smith under whose care she had previously been.

*Physical Examination.*—The patient presented the picture of a small, thin girl, sitting up in a cardiac bed, breathing somewhat rapidly, but in no apparent distress. She was mentally alert and very cooperative. The skull was symmetrical, with no exostoses, irregularities or tenderness; the scalp was clean and without scars, and the hair was dark brown, abundant and fine in texture. The general expression of her face was an expression of alertness, with no indication of discomfort. The skin was normal in color, and the lips were red; there was a slight evidence of cyanosis; the skin was smooth, warm and normally moist, without eruptions or pigmentations. The pupils of the eyes were circular, equal and reacted normally to light and accommodation. The sclerae were bluish white, clear and not jaundiced. There was no photophobia, lacrimation, diplopia, nystagmus, palsies, lid-lag or exophthalmos. The conjunctivae were not remarkable. There was no gross disturbance of vision, and glasses were not worn. The tick of a watch was heard normally from either ear; there was no discharge, stigmas or tophi. In regard to the nose, there were no deformities, obstruction or discharge. The breath was bland, and no ulcerations, exudate or pigmentation were present in the mouth; the lips were red, without herpes, ulcerations or fissures. All the teeth were present, and their condition was good. The gums were normal in color, without pyorrhea, bleeding or leadline. The tongue had a moderately moist coat, protruded in the midline without tremor and was without mucous patches.

The tonsils were absent, there was no exudate. The pharynx was normal in color; there was no catarrh. The uvula was missing. The palate moved normally and there was no evidence of adenoids. The character of the voice was not remarkable, and the thyroid was not palpable. No enlargement of the lymph glands was noted, and there was no pulsation, stiffness or tracheal tug. The thorax was symmetrical; expansion was equal on the two sides, and the back was rounded. The sternum was prominent in front, with marked bulging in the region of the precordium. Respirations were rapid and chiefly costal in type, with a rate of 24. There were no abnormal pulsations. The breasts were not developed, and were without tenderness.

Examination of the heart revealed the apex impulse seen and felt 8 cm. to the left of the midsternal line in the fifth interspace; it was diffuse, heaving and forceful. By percussion, the supracardiac dulness was: 2 cm. in the first space and 4 cm. in the second space; 3.5 cm. to the right of the midsternal line in the third space, 4 cm. in the fourth space and 4 cm. in the fifth space; to the left of the midsternal line, 5.5 cm. in the third space, 9.5 cm. in the fourth space and 11 cm. in the fifth space when measurements were taken around the chest. The heart sounds were regular, rapid and of good quality. The first sound accentuated was followed by a faint blowing systolic murmur. At the apex, immediately following the second sound, was a rumbling diastolic murmur ending in a presystolic crescendo. The murmurs were transmitted to the axilla; a diastolic murmur could be heard at the base, and a long diastolic thrill ended with the first sound. The pulmonic second sound was greater than the aortic second and accentuated. The radial pulses were equal, regular and synchronous, of fair volume and tension, and the vessel walls were not thickened or tortuous; the rate was 112. The blood pressure was: systolic, 90; diastolic, 60.

Examination of the lungs revealed the tactile fremitus normally transmitted throughout. The percussion note was resonant except for slight impairment at both bases, which was more marked on the left. The breath sounds were bronchovesicular in character, except at the bases where expiration was slightly prolonged. A moderate number of moist crackles were heard at both bases. The apexes were clear, and the lower borders moved slightly with inspiration. The abdomen was fairly well developed, symmetrical and level. The percussion note was normally resonant. There were no abnormal pulsations, masses, spasms, hernias or fluid. The abdomen was held somewhat rigidly, voluntarily. Reflexes were present and lively on both sides. Liver dulness extended from the fifth rib to the costal margin by percussion; the edge was not felt. The gallbladder, spleen and kidneys were not felt.

Examination of the lymphatic glands revealed no enlargement of the cervical, axillary, inguinal or epitrochlear glands.

The normal lumbar lordosis was absent, and the vertebrae were prominent in the upper lumbar and lower thoracic region. There was slight upper thoracic kyphosis, with no sharp knuckle, no scoliosis, rigidity or tenderness. The other bones were not remarkable.

There were no involuntary movements of the arms, and no wasting, tremor or clubbed fingers were noted; the nails were not remarkable. Biceps, triceps and radial periosteal reflexes were equal and active on the two sides. No varicosities, scars or ulcers were present in the legs; the knee jerks and Achilles' reflexes were equal and lively on the two sides. There was plantar flexion; no Babinski, Oppenheim, ankle clonus or Kernig's sign were present; the test for Romberg's sign was not made, nor was the patient's gait examined. There was no edema, and the skin reacted normally to the rough test.

*Summary.*—The patient was a girl of 11 who for three years has had increasing shortness of breath. She had been in and out of many hospitals, had not been able to attend school and for the last eight months had been bedridden. Recently, there had been terrifying attacks of hemoptysis. On examination, there were the typical signs of mitral stenosis. The ordinary outlook was hopeless, and, since there was evidence that the musculature of the heart was still in a fairly good condition, operation was dicussed with the patient's parents, who accepted it.

*Laboratory Findings.*—According to the analysis, May 17, the urine was straw colored and acid; the specific gravity was 1.020; albumin and sugar were absent, and the sediment revealed only a few leukocytes.

Examination of the blood, made May 17, revealed; hemoglobin, 85 per cent.; red blood cells, 4,160,000; white blood cells, 8,200; a stained smear showed, polymorphonuclears, 59 per cent.; lymphocytes, 36 per cent.; large mononuclears, 3 per cent.; eosinophils, 2 per cent. The red cells were slightly achromic.

Examination of the stool, May 17, showed that it was normal.

The vital capacity of the lungs, May 17, was 1,100 c.c. (52 per cent. normal). A reexamination made May 18 by the roentgen ray showed marked increase in the hilum shadows and the lung markings near the hilum, suggesting congestion. There was a definite fulness in the region of the left auricle and also the left pulmonary artery. A 7-foot film of the heart showed moderate cardiac enlargement; the measurements were: Midline to right margin, 4.6; midline to left margin, 8.2; supracardiac, 4.5. The internal diameter of the chest was 20.4 cm.

The impression was that of mitral stenosis. The electrocardiographic examination revealed a regular heart, with evidence of right ventricular preponderance and auricular hypertrophy. The Q-R-S waves were sharp and the T waves were upright.

*Operation.*—Cardiotomy and valvulotomy were performed May 20, 1923, for mitral stenosis (Fig. 35), under ether (Connell apparatus). Preliminary morphin, 0.008 gm. and atropin, 0.00045 gm., were given at 7 a. m., and the patient was brought to the operating room at 7:30 a. m., where preliminary roentgenograms were made with the patient in the cardiac bed. The child was extremely upset as

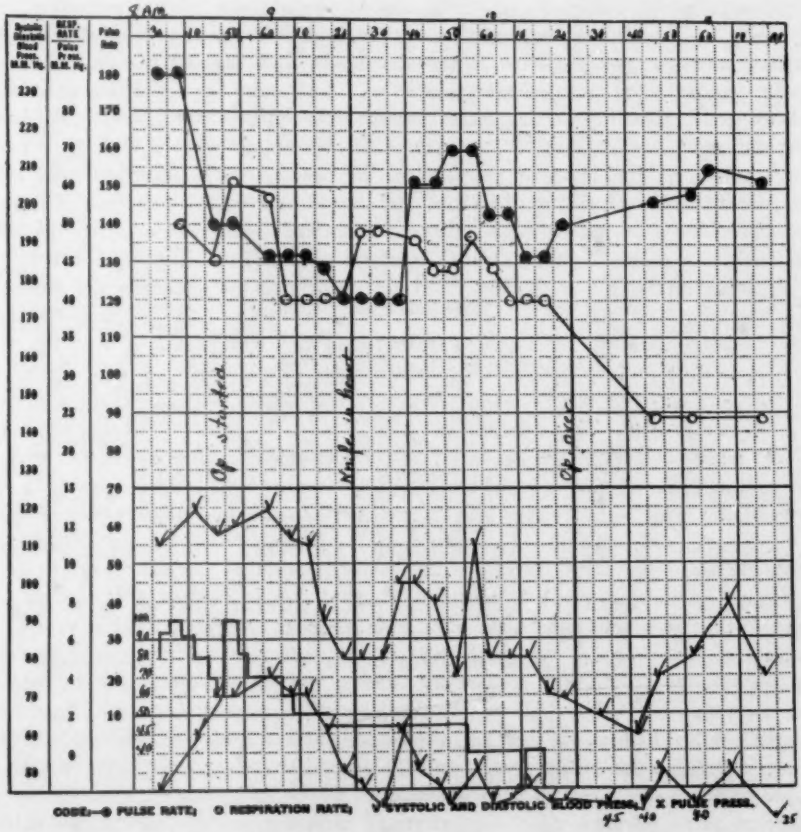


Fig. 35 (Case 1).—Record of pulse, blood pressure, and respiratory rates and ether vapor tension throughout the operation.

soon as the question of anesthesia was introduced, and it was only after the most trying interval that the anesthetic was administered, although it was at once taken easily and satisfactorily and continued as a simple, uninterrupted, quiet anesthesia throughout the operation. The child was then placed on a table inclined at about 45 degrees, a catheter passed into the nasal pharynx through one nostril, and the Connell machine brought into use. At that time, the pulse was 180, respirations 50, and the blood pressure, systolic, 110 and diastolic, 50. Preliminary preparations of the thorax was made with alcohol and bichlorid.

The line of incision was outlined, beginning close to the sternal notch and running down half way between the ensiform and umbilicus. The usual procedure was followed, with towels, screen cover, and a small instrument table over the knees. Opening into the pericardial sac was then performed after the Duval-Barasty median thoracico-abdominal pericardiotomy. The incision, as outlined, was carried fully through to the sternum over the end of the ensiform down to the linea alba. The ensiform was then freed of its attachments to the rectus muscle laterally and the diaphragm posteriorly, after which it was removed. The fingers of the left hand were then inserted beneath the sternum and pushed upwards in the midline, the tissues being swept to each side as progress was made upwards, and the substernal space freed up to the second rib. The ribbon retractor was then laid over the mediastinal contents. It had been planned to perforate the sternum with a burr at the second intercostal space, but because of the softness of the sternum we discarded this idea, which would have been much better for the subsequent closure, and opened the sternum up to the midline to the level of the second interspace with a Horsley osteotome. At the level of the second interspace, the manubrium was then cut across, care being taken to avoid the internal mammary arteries. The two halves of the manubrium were then gently pulled apart, the attached pleurae on each side being swept away from the chest wall as it was elevated. A Tuffier rib spreader was then inserted and widely spread, holding the sternum far apart and making possible a most excellent view into the mediastinum. The pericardium was then picked up between the forceps slightly to the left of the midline and opened up towards the base of the heart, care being taken as the base was reached to avoid the pleurae, as they came into close approximation at this level. This incision was then carried downward to expose the apex of the heart; and, after the opening into the abdominal cavity, which was walled off with a piece of gauze, had been made, the incision was carried towards the crus of the diaphragm splitting the inferior pericardium and diaphragm together.

This provided a beautiful exposure of the heart in full action. The right ventricle was very large and filled the major portion of the exposure; in fact, it rather overlapped the left ventricle, so that it was evident that we should have to tilt the heart towards the right side in order to expose the left ventricle. Since our experimental experience had made us feel that a heart subjected to preliminary gentle trauma will stand sudden and more severe trauma better, the heart was as freely pulled up into the desired position for exposure of the left ventricle several times. It tolerated the amount of handling well, although at this point it became somewhat irregular and seemed to dilate on the right side a little. The blood pressure also dropped to 80 systolic, and it came to be a question as to whether we should continue with the attempt to cut the valve. About 0.5 c.c. of epinephrin was allowed to drop on the heart itself and to spill over into the pericardial sac, with an immediate and excellent response in the action of the heart. This opportunity was seized in which to attempt the desired procedure, and the heart was rolled out with the left hand towards the right side and two deep silk sutures were placed in the heart muscle about one-half an inch apart, well in the center of the left ventricle and away from the coronary branches. The heart was allowed to return to its normal position for a moment; the knife-valvulotome, which we had found very efficacious in dogs, was picked up, and the heart, which was held in the left hand, was again displaced to the right. The valvulotome was then thrust into the left ventricle between the sutures as placed. It was then carried upwards into

the region where we expected to find the mitral valve at the depth of about 2½ inches (6.35 cm.). A firm structure was felt, which we took to be the valve; the knife was then turned mesially, and the structure encountered was divided. It took considerable effort to accomplish this and almost dislodged the heart from the left hand. The knife was then rapidly turned and a short sweep of the blade made in the opposite direction in an attempt to cut the postero-lateral cusp as well. The knife was then carefully withdrawn, and the sutures, which had been previously placed, were tied over the point at which the instrument had been inserted, so that no bleeding resulted. The heart seemed but little disturbed by this manipulation, and if we had had more experience and better courage, another attempt would have been made after a short interval; because, when a sterile stethoscope was placed on the heart it was obvious that the diastolic murmur remained, although there was some definite indication that there was an extension of the systolic murmur, which might be taken to indicate the creation of a greater insufficiency. The pressure remained fair (systolic around 100), but the pulse rate was about 150 and it seemed wisest to retire.

After some warm salt solution had been left in the pericardial sac, the inferior aspect, including pericardium and diaphragm, was first closed with a running stitch of C silk. The opening in the abdomen was then closed in layers with the same material. The anterior pericardial opening was then closed with a running suture of the same material, about 20 c.c. of warm salt solution being left in the pericardial sac as the suture was tied. No definite opening was ever seen in the pleurae, although the lungs, as they were viewed through the thin tissue, seemed to be slightly retracted away. The retractor was then removed, and the two halves of the sternum held firmly together by an encompassing, silver wire suture at the third interspace. The transverse fissure in the sternum was held in position by interrupted multiple stitches of fine silk; many sutures of the same material were placed in the periosteum, covering tissues of the manubrium along the midline opening. Fine silk sutures were applied to the subcutaneous tissues, and silk to the skin; silver foil dressing, swathe and shoulder straps were used. The pressure at the close of the operation had dropped to 80 and the patient seemed a little cyanotic, but the pulse was of a fair quality, and the rate was 145. The patient was placed in a semi-upright position in the heart bed and kept in the operating room until she was thoroughly conscious; the recovery was fairly rapid and without nausea or vomiting.

There was considerable pain felt at once in the region of the incision; it seemed to be produced by a rubbing together of the rough edges of bone. This was only controlled by the use of morphin. By 4 p. m. the patient was more comfortable, and was returned to the ward in good condition.

*Postoperative Notes.*—May 21, the patient was making a stormy operative recovery chiefly because of the pain over the fractured sternum felt on respiration. It was necessary, therefore, to give morphin in 0.010 mg. doses three times in the first twenty-four hours. The pulse remained elevated around from 130 to 140, respirations rose to from 35 to 40 and the child looked cyanotic. Râles could be heard in the apices, particularly in the right; there seemed but little doubt that the patient was suffering from an acute postoperative pulmonary upset. Support of the heart action was given by digitalis by mouth, and oxygen was administered continuously for the cyanosis. The outlook was not bright, although the heart action continued good. The immediate post-operative roentgenogram showed some increase in the size of the heart with

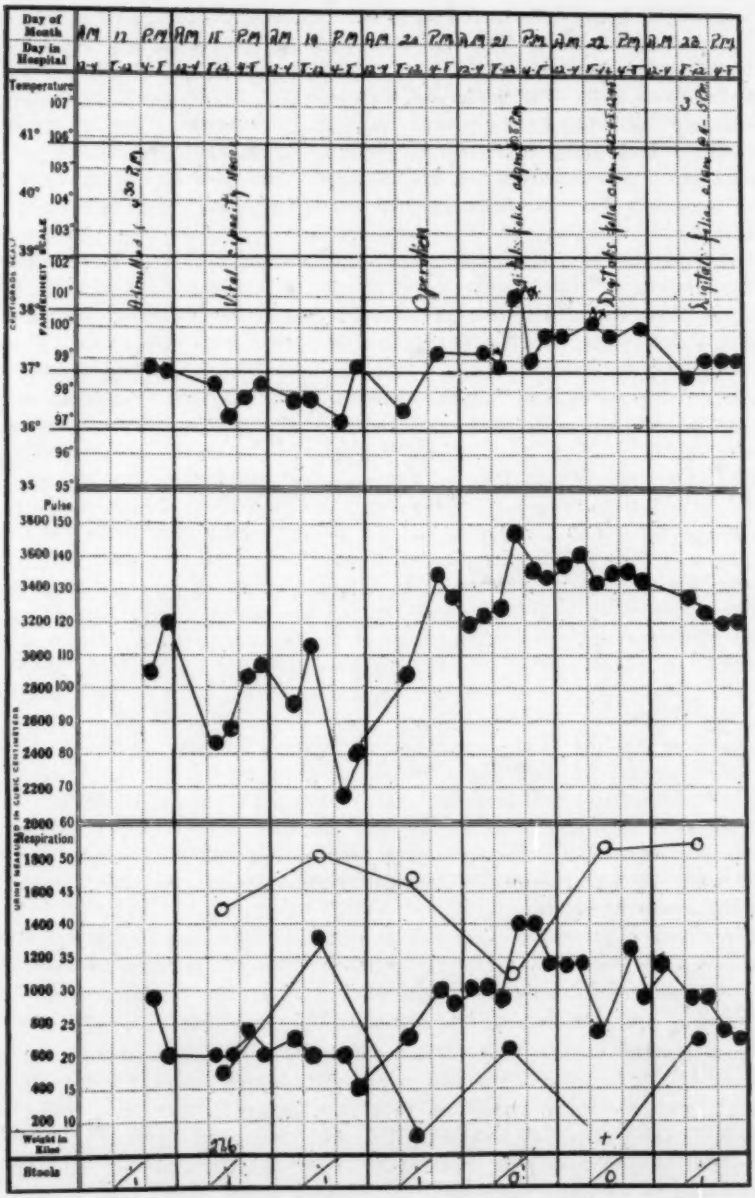


Fig. 36 (Case 1).—Postoperative course for first three days.

some obliteration of the sulcus between auricle and ventricle, which may have been chiefly due to distention of the pericardial sac with salt solution. The patient's condition (May 21) was critical.

By May 24, the pulmonary condition had suddenly cleared up and left the child in apparently good shape. The signs in the right apex, which had indicated bronchial breathing as well as râles two days before, had disappeared. Respirations dropped to 25, the pulse went down to 100, and the temperature to normal (Fig. 36). The general condition of the child appeared excellent; although morphin had been used up to that day to control the pain, it seemed no longer necessary. Continued observation of the heart sounds revealed a possible prolongation of the systolic murmur at the apical region. The bowels had moved naturally, and the child was taking plenty of fluids and voiding spontaneously. Indeed, the condition was so satisfactory that she was brought down to the amphitheater and presented before the reunion of the Brigham Hospital graduates, to whom a brief outline of the experimental work leading up to the operation and the actual operation itself was presented.

The patient's condition, according to the report of May 28, continued uninterruptedly satisfactory. The pulse remained at 100, and was regular, full, and of good quality; the blood pressure was 110; the temperature was normal, and the respirations were about 25. The day before there had been a slight epistaxis from the right nostril, the patient losing perhaps one-half an ounce (14.175 gm.) of blood. The sutures had been removed the previous day, and the wound was perfectly healed. There was no pain now with respiratory movements, and the sternum seems to be healed in good apposition. Ammonium chlorid, which was given to clear up the slight cough that followed the clearing of the pulmonary complication, had been discontinued. The fluid intake was approximately 2,000 c.c.; defecation and urination were normal. A more careful study of the heart sounds was possible at that time, and it revealed a change in the murmurs antedating operative procedure. The chief change was a prolongation of the systolic murmur, heard best in the midaxillary line. The thrill and presystolic rumble were still present. The bony parts had healed in such a way as to leave the chest more prominent than it was before operation, so that it appeared to be of the type of definite chicken breast, which should permit the heart to come forward more.

May 29, the striking thing from a cardiac point of view as to the events during the operation itself was that the heart rhythm was practically not disturbed. The electrical complexes of the heart beat changed their form; probably this change was associated with the manipulation and lack of tone that developed, but no important rhythmic changes occurred. The blood pressure at times was dangerously low, systolic varying between 60 and 40; but applications of hot saline directly applied to the heart or the use of epinephrin proved very efficacious in restoring the strength of the heart beat.

Twenty-four hours after the operation, definite signs of consolidation were discovered at the right apex, that is, bronchial breathing, dulness, bronchophony and râles. This was either a pneumonia or a pulmonary infarct. At that time, the child was desperately ill. Stimulation in the form of digitalis was administered, and the use of oxygen was continued more or less constantly. The latter seemed to be of great value to the patient. The extreme cyanosis was much improved by the oxygen. No careful examination of the heart was made until May 29, because it was not thought wise to disturb the patient too much. At this time, the blood pressure was, systolic 108, and diastolic, 55; no clubbing of the fingers was present. The veins of the neck were not distended; the

pulse was small, and there was questionable slight cyanosis of the lips and finger tips. The sternum was much more prominent than it was before operation. The apex was diffuse, and could be seen and felt in the fourth and fifth interspaces 9 cm. to the left of the midline; the right border, by percussion, was out 5.5 cm. The heart action was regular; the first sound was somewhat accentuated and followed by a moderately loud, systolic blow transmitted to the axilla. At the apex, the entire diastole was filled with a rumbling murmur, with a presystolic accentuation. This murmur was essentially the same as it was before operation, but the systolic murmur was now definitely louder. The pulmonic second sound was accentuated. The thrill felt at the apex seemed distinctly less marked than before operation. To the right of the sternum, in the fourth space, a definite pericardial friction could be heard at the base of the heart. Liver dulness began at the fifth rib, but the edge could not be felt below the costal margin. There was no edema of the legs. The lungs showed diminished breathing sounds and a few râles, together with slight dulness at the right apex front and back. There were also a moderate number of râles at both bases and behind. The roentgenogram, made May 28, which

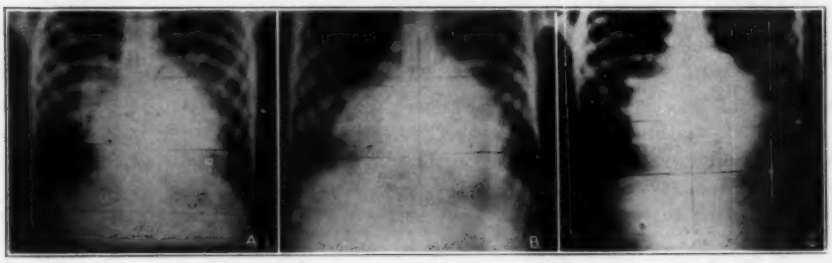

Fig. 37.—Roentgen-ray studies of heart (Case 1). *A* was taken the day before operation; *B*, eight days after operation, and *C*, ten months postoperative. All plates were made under similar conditions of distance (3 feet), time, posture, etc. *A* shows a prominence in the region of the left auricle; *B* shows a great increase in size, apparently due to pericardial effusion, and *C* shows the heart somewhat larger than before operation and peculiarly rounded at its apical portion.

presented an enlarged cardiac shadow, can be interpreted as being due to the presence of a moderate amount of fluid in the pericardium or to stretching of the heart fibers, probably the former. It was considered that, when the lung had cleared up as much as possible, the vital capacity reading would be of considerable value to us in determining whether or not the circulation has been improved by the operation. At that time, the child looked quite comfortable.

*Outcome.*—Repeated roentgen-ray examinations showed that the heart shadow increased markedly for several days (Fig. 37). This was probably due to a pericardial effusion which gradually cleared up. Following this, there was a slow but steady improvement in the general condition of the patient. The vital capacity of the lungs slowly rose to 1,300 c.c., or an increase from 52 per cent. to 61 per cent. of the normal capacity. After several months, she was allowed to get out of bed and walk around. During all this time, there still remained characteristic signs of mitral stenosis; that is, there was a rumbling diastolic murmur with a presystolic crescendo at the apex and a

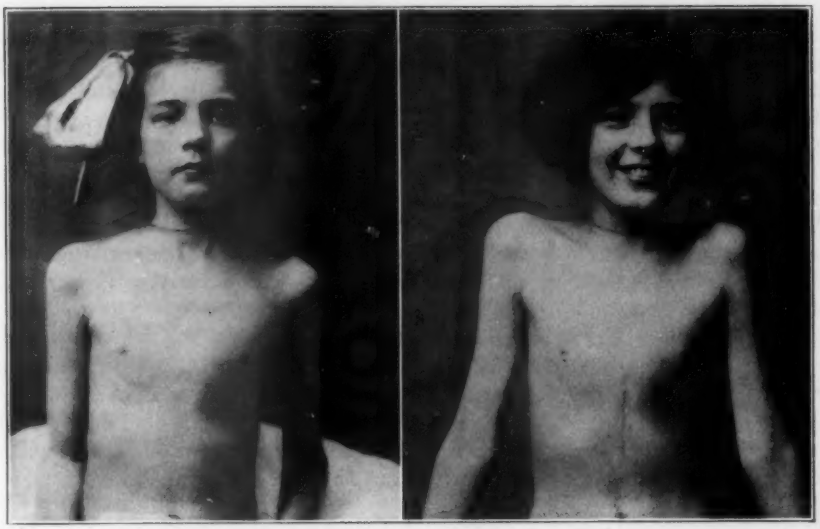

Fig. 38 (Case 1).—Front view before and after operation.

Fig. 39 (Case 1).—Side view before and after operation, the latter showing prominence in the region of the precordium.

presystolic thrill. The only significant change is that, whereas before operation the systolic murmur was barely audible, it became louder after the operation.

About seven months after the operation, the heart rhythm, which had continued regular at a rate of about 100, became absolutely irregular. Electrocardiograms showed that auricular fibrillation had set in. This did not disturb the patient; in fact, she distinctly improved still further, with the change in rhythm and the heart rate at about 80. During all this time there was no edema of the legs or any enlargement of the liver. The patient would stay out of bed all day and walk around. On several occasions, she contracted an infection, had a fever and remained in bed for a short while. Hemoptysis, which was before operation such a distressing symptom, had never recurred since operation. Eleven months after the operation the mother remarked that she thought that her girl was in better condition than she had been at any time in the last few years.[101a]

Figures 38 and 39 are photographs taken before and after operation.

Case 2.—*History.*—S. G. G., a woman, aged 35, single, a dressmaker, born in Russia, was first admitted to the Peter Bent Brigham Hospital, July 24, 1922, with the complaint of palpitation and pain over the breast. The cause of the father's death was not known, or the age at which he died. The mother had died of pneumonia at 30. There were no other children. There was no family history of cancer, diabetes, gout, tuberculosis, hemophilia, arthritis, or diseases of the circulatory, digestive, renal, nervous or muscular systems. The patient had worked hard and worried for the last fifteen years, but work had been given up seven months before because of the present illness. She had lived in New England for the last nineteen years. She had had measles in childhood, and typhoid at the age of 10, but the details were not obtainable, except that she had been very seriously sick and had had severe epistaxis. There was no history of chicken pox, smallpox, diphtheria, mumps, whooping cough, influenza, tonsillitis, chorea, rheumatism, pleurisy, pneumonia, malaria, or scarlet fever, and there was no association with tuberculosis. There was no history of injuries and operations. There was no history of headache or trauma; glasses were not worn, and there was no failing of the vision, inflammation or pain. There had been no loss of hearing or any pain or discharge in the ears. Head colds had been rare before the onset of the present illness; there was no discharge, epistaxis or any symptoms of obstruction. The patient had always taken good care of her teeth; some dental work had been done two years before.

There had been no tonsillitis, attacks of sore throats, hoarseness or sore mouth. There was no pain in the chest, palpitation, or dyspnea previous to the present illness, as well as no cough, sputum, hemoptysis, or night sweats.

The patient's habits of eating were good; the bowels had always been constipated, and cathartics were used daily until she entered the hospital. Previous to the present illness, there had been no nausea, gas or vomiting, no hematemesis, distress, colic, icterus, diarrhea, bloody, tarry, or clay colored stools; because of constipation she had had bleeding hemorrhoids for many years. There was no history of nocturia, dysuria, hematuria, pyuria, or smoky urine, and retention or incontinence had not been experienced. Three years before admission the patient was ill with severe, sharp pain in the back, and while she was at the Baptist Hospital a stone was passed, which was diagnosed as a kidney stone. There had been no return of symptoms. There was no history or any symptoms of venereal disease.

The menses first appeared at the age of 16, occurring regularly every twenty-four days, and lasting four days; the flow was moderate in amount, and no pain was present. There was no leukorrhea. The last period had occurred two weeks previous to admission. For years she had been unable to sleep well; she was very much depressed, and cried often. No vertigo, fainting, twitching spasms, anesthesia, paresthesia, ataxia, girdle, shooting, muscular or joint pains were present, and the memory and disposition of the patient were good. Before the present illness her weight was 130 pounds (59 kg.). It had varied since then, and on admission was 107 pounds (48.6 kg.).

About ten years previous to her admission, the patient was told that she had a weak heart. Before admission she had first noticed that her heart "pounded" unduly when she was dancing. There was no shortness of breath at this time, but such vigorous exercise was given up. The winter before admission she had had frequent bad colds, as well as dysmenorrhea, with increased frequency of periods (two weeks), excessive flow and considerable pain. Seven months before she had begun to have attacks of palpitation, with nausea and vomiting not associated with exercise. She was unable to work. There was swelling of the ankles, questionable ascites and enlargement of the liver. After that, she had had a series of hospital admissions, and during this time her symptoms would subside only to return again shortly after she returned to work. She has had no cough or orthopnea.

The patient was discharged from the hospital, September 8, her case having been diagnosed as valvular disease, combined chronic, cardiac, mitral insufficiency and stenosis, and auricular fibrillation.

She was readmitted to the hospital, Oct. 4, 1923. Since her discharge from the hospital, the patient's condition has not changed appreciatively either symptomatically or subjectively. She has never been free from palpitation and pain over the heart region, and for the last seven or eight months had been very short of breath. No work of a strenuous nature had been done. For the last three months the patient was at the Baptist Hospital under observation by Dr. J. H. Pratt. She had felt quite comfortable, and was entering the hospital for the purpose of securing surgical intervention in the hope of relieving her serious cardiac condition.

*Physical Examination.*—The patient presented the picture of a well developed and well nourished young woman aged 35, resting upright in bed, in no pain, but able to talk and to get her breath with difficulty. The skin was smooth, moist, pale, and elastic, with no eruptions or pigmentations. The head was well-formed and symmetrical, with no exostoses, scars or tenderness. The pupils of the eyes were equal, regular, and reacted to light and accommodation. The extra-ocular movements were normal, and there was no strabismus, ptosis or exophthalmos. The reaction of the ears was negative as regards mastoid tenderness, tophi and discharge, and there was no obstruction, discharge or bony deformity of the nose. The tongue was clean, moist, and protruded in the midline without tremor. No teeth were missing. There were twelve gold fillings. Moderate pyorrhea was present, but no leadline. The tonsils and pharynx were not injected.

The thyroid was not palpable, but there were definite visible and palpable pulsations of the veins of the neck. There was no enlargement of the glands in the neck, axillae or groins. The thorax was well-formed and symmetrical; expansion was good and equal on the two sides. Normal resonance was present throughout the lungs, and the breath sounds were vesicular in quality. No râles were found.

There was a visible heaving impulse of the heart over the whole left chest. The apex was forceful, and was seen and felt 14.5 cm. to the left of the midsternal line. The left border of dulness was located 15 cm. to the left of the midline; the right border, 5 cm. to the right. The heart action was slow and grossly irregular; the first sound was not accentuated, but there was a soft, small systolic roll, heard best at the apex and in the third left interspace. The second sound was followed by a long, diastolic murmur. There was a definite thrill at the apex, questionable in time. The second pulmonic was greater than the aortic closing. The radial pulses were equal, absolutely irregular, with a rate of 90. Tension and volume were good, and there was no pulse deficit. The walls were easily collapsible and not thickened. The blood pressure was: systolic, 136; diastolic, 86.

The abdomen was level, soft and tympanitic throughout. There was no spasm, masses or tenderness. The liver edge, the spleen and the kidneys were not palpable, and there was no costovertebral tenderness. The extremities were equal in length and well-formed. There was no edema, tremor, clubbing varicosities, scars or ulcers. The knee jerks were active and equal, and there was no Babinski's reflex, ankle clonus or Kernig's sign. Rectal and pelvic examinations were not made.

*Summary.*—A woman of 36 had been suffering from increasing shortness of breath and precordial pain for over four years. She had been told that she had heart trouble about eleven years before. During the last 1½ years previous to her admission, she had been in hospitals five different times, each time improving but not able to do any work whatever in the intervals. She developed edema of the legs, enlarged liver and, on examination, showed signs of mitral stenosis and auricular fibrillation.

*Laboratory Findings.*—According to an examination made October 15, the urine was amber and acid, with a specific gravity of 1.020; there was the slightest possible trace of albumin, and sugar was absent. The sediment showed no casts or blood. The blood examination (Oct. 5) revealed: white blood cells, 7,600; hemoglobin, 70 per cent. October 6, the examination revealed: red blood cells, 4,544,000; a smeared stain showed moderate achromia. The white blood count and platelets appeared normal. There were: polymorphonuclears, 80 per cent.; lymphocytes, 16 per cent., and mononuclears, 4 per cent. The phenolsulphonephthalein excretion, October 5, was 50 per cent., 200 c.c. water having been used.

A 7-foot film of the heart, made October 5, revealed considerable enlargement in the region of the left ventricle. The left auricle was more prominent than usual, and the left pulmonary artery was more definite. The findings suggested a mitral disease. Measurements of the heart were: midline to right margin, 4.4; midline to left margin, 11.5; great vessels, 5.9. The internal diameter of the chest was 25 cm. Stereoscopic films of the chest showed an increase in the hilum shadows and lung markings, which was rather generalized, suggesting congestion.

*Special Note.*—October 5, the patient lay in a semirecumbent position, fairly comfortable. The blood pressure was: systolic, 116; diastolic, 76; the vital capacity of the lungs was 2,200 c.c. and 2,275 c.c.; the weight was 49.4 kg. There was practically no tonsillar tissue; she had never had tonsillectomy done.

The right border of the heart measured 3.8 to the right in the third space, and 4.5 to the right in the fourth space. The apex impulse on the left border was 16 cm. to the left of the midline; the apex rate was 71; its action was absolutely irregular. The first sound at the apex was diminished in intensity, and was followed by a moderate systolic murmur. There was a definite, faint, early, diastolic murmur coming a short interval after the second sound. The aortic second was definitely diminished, and the pulmonic second was accentuated. Both systolic and diastolic murmurs were best heard in the fourth left space near the sternum; no definite thrill could be felt, although a slow vibration was distinguished at the apex. The upper border of the heart, by percussion, was felt over the third rib. There was no pitting edema of the legs. The liver was a hand's breadth below the ensiform, not tender, and the edge was not felt, although the surface seemed to be definitely made out. On the morning of October 5, there were no râles at the base of the lungs, but the right base was somewhat dull, probably due to a high liver. There was slight cyanosis of the lips. The general condition of the patient seemed fairly satisfactory.

The diagnosis of mitral stenosis and auricular fibrillation was made. The patient seemed to be in as good condition as possible, and from her story it was perfectly evident that she would not be able to increase her activities on getting out of bed. This had been repeatedly tried, and each time cardiac failure would develop, with edema, dyspnea and a rapid heart. Her heart muscle seemed still to be in good shape, and there seemed to be a serious mechanical difficulty in the mitral valve which prevented her from doing more. Judging from these facts, she was a suitable subject for surgery of the mitral valve. It was not considered that there would be any unusual difficulties. She was not given digitalis because of the slow heart action, and because a very short time before, while Dr. Pratt was administering digitalis, she had showed coupled rhythm.

*Operation.*—Oct. 7, 1923, valvulotomy was performed for mitral stenosis (Fig. 40), under ether (Connell apparatus). The patient was given 10 mg. morphin and 0.0005 gm. atropin hypodermically at 7 a. m. and brought to the operating room at 7:45 a. m. At that time, the blood pressure was 85, and the pulse was approximately around 120 or 130. She seemed, however, to be in as good shape as we would probably ever find her. Ether was administered by means of the open mask; it was well taken. It was then transferred to the Connell machine, and proceeded smoothly and most satisfactorily throughout the operation. The usual preparation with alcohol and bichlorid was made.

A midline incision from the sternal notch to a point 3 inches (7.62 cm.) above the umbilicus was made; this incision was carried through to the sternum and through the linea alba, the peritoneum not being opened at the lower angle of the wound. The attachment of the rectus was then freed from the ensiform, the ensiform removed with the rongeur, the fingers inserted below the manubrium and the tissues posteriorly swept away from the manubrium to a point as high as possible. Two holes were then burred in the center of the manubrium, one at the level of the second interspace, and one half way between this and the tip. These were enlarged, a Gigli saw guide was passed beneath the manubrium, pulled through these burr holes, and the sternum cut across with the Gigli saw. The two halves of the sternum were then gently pulled apart, the pleura on either side being wiped away with wet pledgets of cotton. A tear occurred at the upper limit into the right pleural cavity, but the lung was easily held distended by the pressure of the Connell machine and the defect was soon repaired with fine silk. The Tuffier retractor was first used

to hold the manubrium open. Then, the anterior pericardium was opened well up to the base of the heart, the precordium was opened widely and the diaphragm split towards the crus. When the diaphragm had been split, the peritoneum also was opened for a short way, the Tuffier rib spreader was substituted for Lilienthal's rib spreader and the two halves of the sternum widely opened, giving about an 8-inch (20.32 cm.) opening, with a splendid view of the heart in full action. In the lower angle of the wound, a very large, engorged, blue liver was seen. The abdominal cavity was walled off with wet gauze and the heart observed.

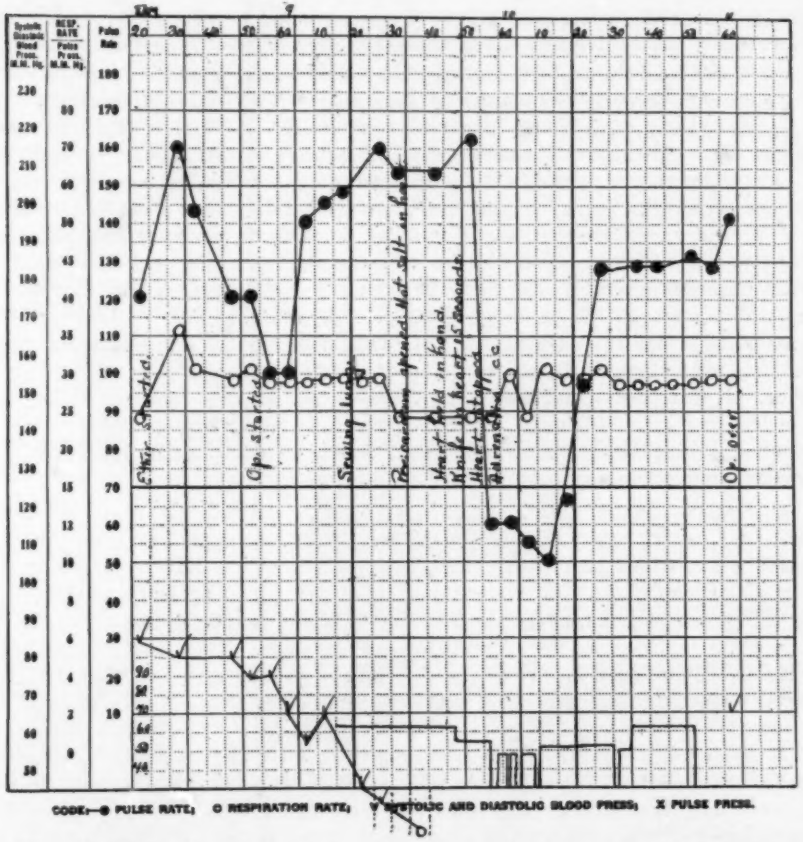


Fig. 40 (Case 2).—Record of pulse and respiratory rates, blood pressure, and ether vapor tension throughout the operation.

Observation of the heart proved most interesting. The right auricle was enormously dilated, blue in color, and in complete rest, without even fibrillary motions running across it. The right auricular appendage from time to time showed irregular convulsive contractions. The right auricular appendix had the appearance of being solid with clot. The right ventricle was of fair size and rather pale, but the descending branches of the coronary vessels appeared to be in a fair condition. The left side could only be observed when the heart was entirely rotated out of its position. This was done several times with the

left hand, the apex being pulled anteriorly and mesially. When this was done, a dilated left auricle was exposed and seen, and the left auricular appendix could be seen peeking around the corner posteriorly in a most inaccessible area. The heart action, of course, remained irregular, just as it had remained for years, and the auricles throughout showed no tendency to contract; presumably, they had been in a fibrillating condition for several years at least, according to the history and to previous observations. We were confronted with a heart in a worse condition than we had anticipated, and it might have been wiser to have retracted our steps then; but the disappointment to the patient and the hope of future life made it seem essential to continue. With gloves removed, which enabled us to handle the heart with more facility, we took the knife-valvulotome which was so successfully used in the case of animals and, rolling the heart out of its position with the left hand, inserted it into the left ventricle rather near the apex and pushed upwards. The instrument entered a large cavity, but it could not reach the valve because the knife handle was too short. It was necessary to withdraw this knife and put a suture in the opening. A larger tenotomy knife, which we had adopted for this purpose, was then used, and the heart was again rolled out of its position, its action appearing to be but little disturbed by the previous manipulation. The knife was then passed upwards into the region of the orifice. What seemed to be the orifice was felt, and the edge of this was cut in both directions. We were not at all certain that we had cut the obstructing ring, because there was not the necessary thickness or toughness to the structure that was felt. The knife, however, was withdrawn and a suture placed to control bleeding. Feeling now that, having gone thus far, we might as well be absolutely certain, and wishing to have better control of the heart, a silk suture was placed in the apex for traction. The assistants retracted these mesially in order to pull the apex well into view, thus allowing the operator to feel with the left hand as well as helping to hold the heart in the new position. The long knife was again inserted, this time still nearer the region of the coronary sulcus, perhaps half way from the apex into the left ventricle, and passed upwards along the posterior wall of the left ventricle where a definite structure was felt. This was cut with the knife. It was thought to be the posterolateral cusp, and the cut was directed toward what should have been the middle of the valve. After a certain distance had been incised, a hard, calcareous area was encountered. This, in turn, was cut across with some effort, the effort tearing an aperture in the ventricle through which the knife had been passed. It was necessary to retire from the field at once, but the bleeding was controlled with little difficulty, a single silk suture sufficing. The heart momentarily stopped beating. One half cubic centimeter of a 1:1,000 solution of epinephrin was dropped onto the heart; this was followed by cardiac contractions which began, interestingly enough, at the rate of about 50, whereas the previous rate had been about 165. This new rate was continued for some time, but it gradually increased throughout the rest of the operation. After an interval, the heart was massaged as it was thought that the contractions were insufficient, but it seemed to make very little difference in the action of the ventricles. Meanwhile, the patient's color continued fair, although the blood pressure was so low that readings were only obtained at rare intervals, and throughout the latter part of the operation they were not obtained at all. Closure was made as in the previous case, and the posterior pericardium was first closed with a running suture of fine silk. The pericardial cavity was filled with a warm salt solution and the anterior pericardium closed again with a running suture of fine silk. The split in the

diaphragm was closed with another running suture of the same material; interrupted No. 2 chromic catgut was used to close the midline aponeurosis of the abdominal wall, and two silver wire ligatures were passed about the two halves of the sternum, one in the region of the fourth interspace and one at the seventh interspace. When pulled tight, this gave good approximation. An interrupted layer of fine silk sutures was placed in the subcutaneous tissue throughout the entire length of the wound, silk to the skin, and swathe; immediate roentgenograms were taken. The patient seemed to be in fair condition at the close of the operation, with a heart rate around 140, and a pressure of 65. She was placed in the cardiac bed and control roentgenograms were taken.

*Outcome.*—The condition of the patient, Oct. 7, 1923, remained more or less the same throughout the day, the pulse varying from 140, soon after the operation, to 120, shortly before death. The patient was given 10 mg. morphin shortly after the operation; 250 c.c. salt solution was given per rectum after the operation and again at 4 p. m. This was retained. The patient was kept warm with hot water bottles, and by 4 a. m. she was perspiring freely, felt warm and seemed to be in good condition. At 4:15, she recognized us, and we felt that the situation was really satisfactory. However, the blood pressure began to fall, and the pulse could not be felt much after 7 p. m. The nurse noted that the respirations became stertorous, more labored and more rapid, and that the pulse at the neck was much weaker. At 8 p. m. rectal saline was given, and the patient's condition was apparently worse at 8:25 p. m. when she seemed to be in extremely critical condition, although the nurse had stated that only a little while before she was in a much better condition. The patient, although she was breathing quite regularly, gave a sudden gasp and died almost at once, at 8:30 p. m. (Fig. 41).

We do not think that we can state definitely the cause of death. Of course, the mechanical interference of the heart was great. It is most probable that the myocardium could not stand the added trauma and burden thrown on it. At the same time, the sudden cessation at the very last may have been evidence of an embolus, either cranial or pulmonary; both of these might be expected when it is known, as we knew it, the nature of the auricular mechanism.

Many lessons were learned. We feel, even more strongly than ever before, that an approach to the auricle will never be feasible. Next in importance was the definite impression that one should be able either to calibrate or locate in some better fashion the condition of the valve before operation as well as at operation. Whether an endoscope can be used through the ventricular wall is doubtful, but perhaps this can be perfected.

*Necropsy.*—The heart, removed in the pathologic department three days previous to this note and examination (Oct. 11, 1923), was washed out with salt solution and filled with formaldehyd solution, the apertures of the veins and vessels being tied off after injection. The entire organ was preserved in formaldehyd. We felt at this time that the heart was sufficiently hardened to examine it. First the left auricle was explored by a right angle incision that gave us a trap-door arrangement through which to look into the heart and yet which might be replaced, for future photography, etc. (photographs of the heart were taken before any manipulations were made). Looking into the left auricle, we observed that there was a tiny clot in the left auricular appendage, that a small portion of this steamed down through the mitral valve and that, in the region of the mitral valve, lay a larger clot, almost 1 cm. in diameter. We later found, on opening the ventricle, that this clot extended down to

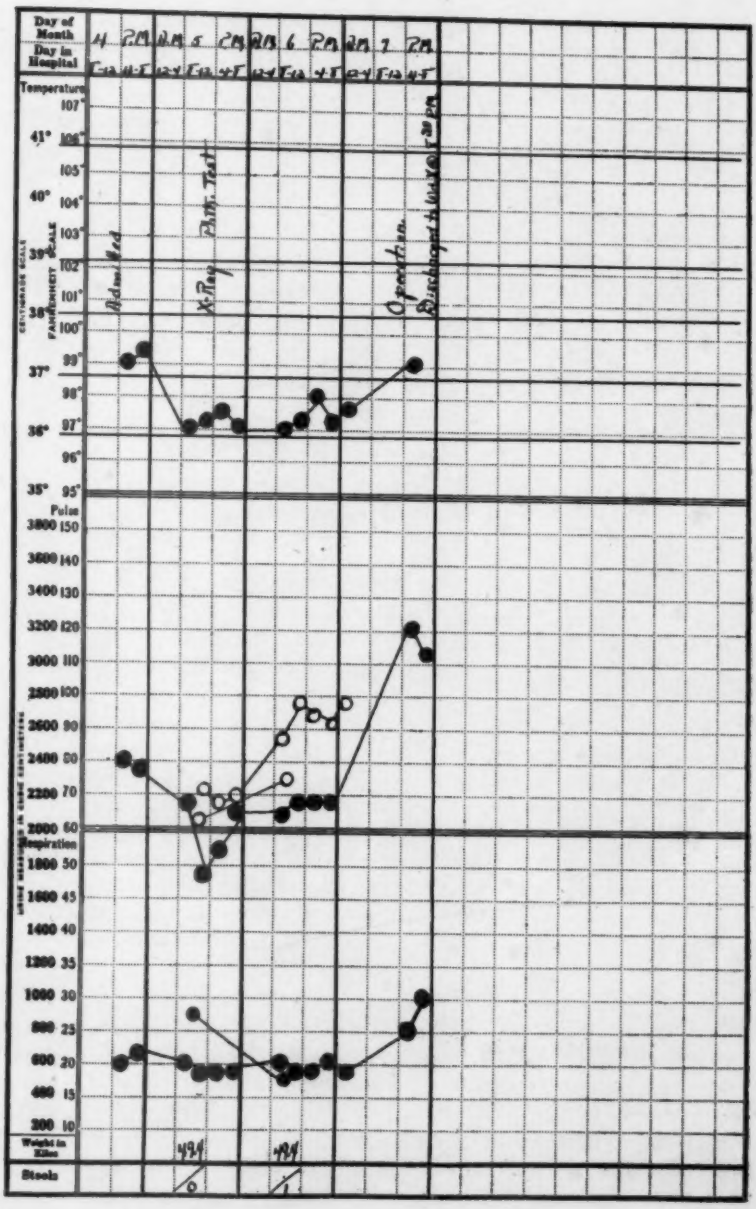


Fig. 41 (Case 2).—Clinical chart.

the papillary muscles almost to the apical region. It was impossible then to decide whether this was an antemortem or postmortem clot. There was a typical, mitral, stenotic lesion, the valve being puckered to a small central opening with calcified edges, rolled on itself and greatly thickened. As could be seen from above, one of the incisions made with the knife had definitely

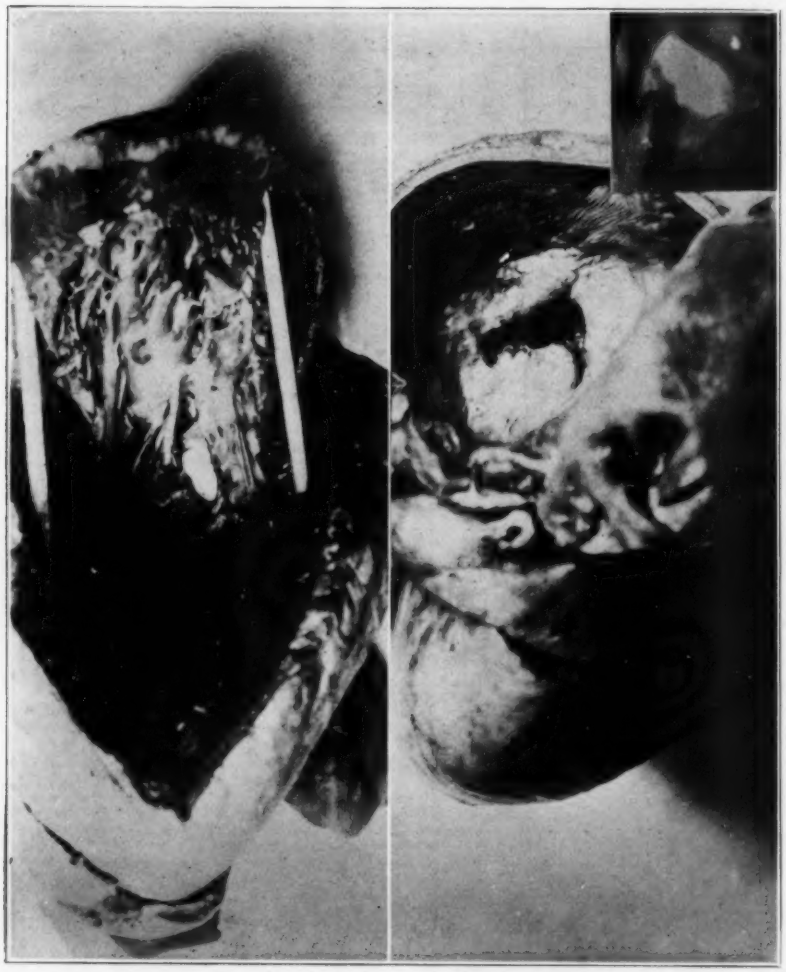

Fig. 42 (Case 2).—Auricular and ventricular views, mitral orifice; roentgen-ray film (inset) shows some calcium deposition. Sharp border of valve edge in ventricular view where incision was made at operation may be noted.

increased the size of this opening (Figs. 42 and 43). The left ventricle was then opened with a V-shaped incision preserving the papillary muscle attachments, and we could look at the mitral valve from below. There was a small, superficial clot caught in the papillary muscles posteriorly, close to the point of entrance of the knife thrust from outside. It was quite thin, paper-like and of

little density. One could see the larger clot mentioned above, which extended through the mitral orifice and extended down towards the papillary muscles again. It was irregular and thick, in some places possibly from 0.5 to 1 cm. thick. This clot was gently pulled down into the ventricle, a tiny, connecting link in the left auricle between valve and auricular appendage breaking off. It was then obvious that we had very successfully obtained an increase in the size of the mitral opening, because it was shown that what had previously been a flattened oval had, by the cut of the knife (most assuredly the last cut made), widened the opening to a triangular orifice; and the sharp glistening edge, as evidence of the knife cut, was very clear. A small, right angled, trap-door incision was made in the right auricle, disclosing the fact that no tricuspid stenosis existed. Drawings were made of the heart with the clot in position, with the artist looking at the opening from below; a drawing was also made with the clot removed. It was generally thought that the clot described above

Fig. 43 (Case 2).—Mitral orifice, ventricular aspect; the incision of the mitral ring increased the aperture and allowed the orifice to become oval rather than slit-like.

was antemortem in its formation, and that it had its origin in the mural thrombus in the left auricular appendix.

Case 3.—*History.*—R. J. S., aged 26, a machinist's helper, born in Massachusetts, was admitted to the Peter Bent Brigham Hospital, Dec. 28, 1923, complaining of shortness of breath, and pain in the chest. The father, aged 53, was alive and well except for a disability which was the result of a fall. The mother had died at the age of 30 from an unknown cause. One brother, aged 28, died, after an accident. One sister was living and well at the age of 30. Except as already indicated, there was no family history of cancer, diabetes, gout, tuberculosis, hemophilia, arthritis or diseases of the circulatory, respiratory, digestive, renal, nervous or muscular systems. The patient lived with his father, his sister and her husband in a five room flat, one flight up. He had no dependents, but was dependent on his brother-in-law, as was his father

also. The patient did not need to return to work because of financial conditions, although he would have liked to return. The rent was being paid, while he was in the hospital. He had a home suitable for convalescence, and there were no language difficulties. Financial worries were not pressing. His brother-in-law made enough money, but there was considerable internal dissention and strife in the family, because his father was also disabled, and the two did not get along well together. Medical care prior to the present illness had been adequate. The patient had not had to work while he was sick. He was probably intelligent enough to follow diet instructions, to pay for the same, and to rest at home when it was necessary.

The patient was accustomed to get about eight hours sleep at night, and he took very little exercise. During the present illness, he had not been working, and spent a great deal of time resting in bed; the rest of the time he spent in reading and writing, which were his only interests in life. He did not use drugs, and he was limited to one cup each of tea and coffee a day. He drank a great deal of milk, used no alcohol, and smoked a pipe, two or three pipefuls a day. He did not use morphin, veronal or patent medicines. The patient had had rheumatic fever at the age of 6, but no chorea, pneumonia or typhoid fever. Previous hospital entries had been made at the Boston City Hospital, thirteen years before when the patient had diphtheria, and, a few days later, scarlet fever. He was a patient at the Boston City Hospital twenty years before, when tonsils and adenoids were removed under ether. There was no history of injuries.

At the age of 14, following a running race, in which he overexerted himself and injured his knee, he developed rheumatism of both knees. The knee joints were considerably swollen but were not painful to a marked degree, although the patient states that they kept him in bed for three years, practically continuously. He had had no trouble with his knees since; no other joints were then involved, nor have they been involved at any other time. He had done heavy work until one and one-half years before his admission, when he suddenly developed a pain in the chest, stabbing in nature, just under the sternum, and on both sides. It was more pronounced in the left side. It came on suddenly when he was engaging in heavy work and lasted three weeks; during this time he had dyspnea and pain, and remained in bed. He got up gradually day by day, but was unable to do work after that, because he felt weak. After that, he remained in the house most of the time, reading and writing and going out very little.

He came to the Brigham Hospital (out-patient department), where he saw Dr. George Denny, and he had been coming more or less regularly every month, to be looked over by him. The last time was the date of admission, when he came with his father, as usual, although he was not feeling very well. Dr. Denny advised him to enter the hospital for a rest.

While going up the stairs of the out-patient department he experienced another attack of pain, sharp in character, and situated over the ensiform cartilage. This pain continued to bother him, but it was not very severe.

During the last one and one-half years previous to admission he had been somewhat short of breath on exertion, but he had never noticed any swelling of the legs or ankles.

*Physical Examination.*—The patient was a fairly well-developed and well-nourished man, lying in bed with a very slight dyspnea. His mental state was clear. The skull was symmetrical, with no exostoses, irregularities or areas of tenderness. The scalp was clean and there were no scars. The hair was

reddish, of moderate texture and moderately abundant. The general appearance of the face was not remarkable except for the presence of freckles. The skin was pale and considerably freckled throughout; it was normal in texture and temperature, and without eruptions or scaling. The pupils of the eyes were equal and regular, and reacted well to light and accommodation. The sclerae were pale, and there was no photophobia, lacrimation, diplopia, nystagmus, palsies, lid-lag or exophthalmos. The conjunctivae were pale. There were no gross disturbances of vision, and no glasses were worn. An ophthalmoscopic examination was not made. The ticking of a watch was not heard at all in the right ear, but it was heard at a normal distance from the left ear. There was some discharge from the right ear, but the left showed normal appearance. There were no tophi. No deformities, obstruction or discharge were present in the nose. The breath was bland, and the mouth contained no ulcerations, exudate or pigmentation. The lips were slightly cyanotic, with no herpes, ulcerations or fissures. Two or three of the teeth were missing; a good deal of dental work had been done, and there were some gold caps and a few cavities. Normal redness was present in the gums, with slight pyorrhea; there was no bleeding, and no leadline. The tongue was slightly coated; it protruded in the midline without tremor, and there were no mucous patches. There was no exudate in the tonsils. The pharynx was normal in color and without evidence of catarrh. Palate reflexes were obtained; adenoids were not examined. The character of the voice was not remarkable. The thyroid was not palpable. There were no bruits, thrills, pulsations, stiffness or tracheal tug; the lymph glands were not palpable. The thorax was symmetrical, expanded well on both sides, and was normal in size and shape. Respirations were normal in rate, depth and character, and no abnormal pulsations were present.

The apex impulse of the heart was seen and felt 11.5 cm. to the left of the midsternal line, in the sixth interspace; it was rather forceful in character. By percussion, the left border of dulness was located 12.5 cm. from the midsternal line in the sixth interspace. The right border of dulness was 3.5 cm. in the third and fourth interspaces. On auscultation at the apex, the first sound was very loud and snapping in character. The second sound was relatively distant, followed by a soft, blowing murmur, fading for an instant in mid-diastole, and becoming loud and forceful just before the first sound, into which it merged. A very slight, systolic murmur was distinguished. Over the base of the heart, there were corresponding murmurs heard, though much less distinctly. The aortic second sound was rather faint, but the pulmonic second was loud, snapping, and somewhat prolonged. The sounds were regular in rhythm. A presystolic thrill was felt on palpation, at the apex and extending up as far as the third rib on the left. There was a palpable, sharp, second sound in the pulmonary area. In the aorta, there was supracardiac dulness in the first space 6.5 cm., and in the second space 6.5 cm. Over this area, murmurs were heard, which corresponded with murmurs heard at the apex but were less loud. The radial pulses were rapid and equal, and regular in rhythm and rate, and the vessel walls were not thickened. The veins were not abnormal. The blood pressure was: systolic, 108; diastolic, 80. The lungs were resonant on percussion; on auscultation, breath sounds were found to be vesicular in character. No râles were discovered. The tactile fremitus was normal. The lower borders moved well with respiration.

The abdomen was well-developed and symmetrical, with no visible peristalsis, no tenderness, no abnormal pulsations, no masses, hernias or fluid. There was a considerable amount of voluntary spasm. It was resonant on

percussion, and moved well with respiration. Reflexes were not obtained. The liver extended, by percussion, to the costal margin, and its edge was not felt. The gallbladder, spleen and kidneys were not palpable. The hernial rings were not enlarged. The lymphatic glands were not enlarged in the neck, axillae, groins or epithrochlear regions. The spine showed no scoliosis, lordosis, rigidity or tenderness; the other bones were not remarkable. The arms showed no involuntary movements, wasting or tremor. There was slight clubbing of the fingers. The nails were not remarkable. Biceps, triceps and radial periosteal reflexes were obtained and found to be equal on the two sides. There were no joint changes. The legs showed no varicosities, scars or ulcers, and the knee jerks were equal and active. The Achilles' reflexes were obtained with difficulty on both sides. There was plantar flexion. No Babinski's reflex, no Oppenheim's sign, ankle clonus or Kernig's sign were present. Test was not made for Romberg's sign, and the gait was not observed. No edema and no joint changes were present. There was no anesthesia, paresthesia or hyperesthesia.

*Summary.*—A man of 26 was unable to work for one and one-half years because of dyspnea and precordial pain. He was confined to his home and sometimes to his bed during most of this period. On examination, an enlarged heart and signs of compensated mitral stenosis were revealed. It was thought that the mechanical embarrassment was considerable and the muscle strength satisfactory.

*Laboratory Findings.*—According to an examination made December 28, the urine was amber and neutral; its specific gravity was 1.020; albumin and sugar were absent, and the sediment contained no casts, blood or pus. An examination of the blood, made December 28, revealed: hemoglobin, 100 per cent.; red cells, 5,328,000; white cells, 15,700. A stained smear showed: polymorphonuclears, 77 per cent.; lymphocytes, 20 per cent.; large mononuclears, 3 per cent.; the appearance of the cells was normal. On December 30, there were 10,700 white cells. The Wassermann reaction was negative. The vital capacity of the lungs was 2,100 c.c., or 72 per cent. of the normal capacity (the height was 155 cm.; weight, 48.8 kg.; surface area, 1.46 square meters; calculated, normal, vital capacity, 2,920 c.c.).

A 7-foot film of the heart made January 3, showed moderate cardiac enlargement, with marked prominence of the left auricle and the left pulmonary artery. The findings indicated a mitral disease, with probably a congenital lesion in addition. The lungs showed considerable congestion. The measurements were: Midline to right margin, 5.2; midline to left margin, 9.6; great vessels, 5.7. The internal diameter of the chest was 25.6 cm. The electrocardiographic examination, December 31, revealed a left ventricular preponderance.

*Subsequent Preoperative Findings.*—The patient had no fever; the pulse was 90 at the beginning, but fell at times to about 75. He lost 3 kg. as a result of diuresis, having received a fair amount of digitalis. He seemed to be in a satisfactory condition, although he knew that shortly after getting out of bed and walking around he would be troubled with shortness of breath and precordial pain. Realizing his limitations and the dangers accompanying an operation, he consented to the operation after the entire situation was thoroughly discussed with him.

*Operation.*—January 12, cardiotomy and valvulotomy were performed for mitral stenosis (Fig. 44), and lysis for adherent pericardium under ether (Connell apparatus). A preliminary injection of morphin, 10 mg., and atropin, 0.0001 mg., was given. The patient was bright and cheerful the morning of the operation. The anesthesia was well taken to begin with, although some difficulty was experienced in keeping him under sufficiently in the early stages of the operation because of failure of the Connell machine to work well. The blood pressure, however, remained at a fair rate throughout most of the operation, dipping down to 60 now and then from 90 and 110. The usual alcohol and

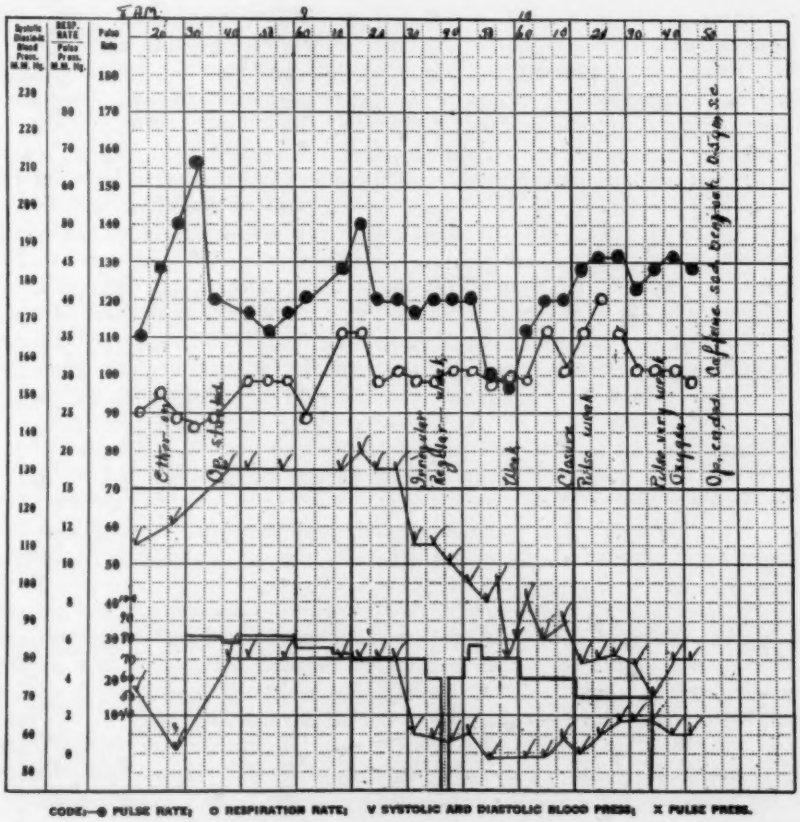


Fig. 44 (Case 3).—Record of pulse and respiratory rates, blood pressure and ether vapor tension throughout operation.

bichlorid preparation of the thorax and upper abdomen was made. A midline incision was made from the sternal notch to a few inches above the umbilicus, carried through the bone of the sternum and through the linea alba in the abdomen. The ensiform, freed of its attachment to the rectus muscle, was removed with the rongeur. The fingers of the right hand were placed beneath the sternum and the pericardium, and the lungs were pushed away from its lower surface. The Horsley bone forceps were then used to split the sternum up the midline to the level of the second interspace, where on either side the sternum was cut across. A Lilienthal rib-spreader was then placed in the

wound, and the ribs opened up widely. The pleurae were pushed to each side. Good exposure was obtained. The pericardium was then opened into anteriorly, disclosing to our surprise the fact that everywhere were old adhesions some delicate and a few very dense between the pericardium and the heart. The diaphragm and the inferior pericardium were then split towards the crus. The adhesions existed, for the greater part, anteriorly and in the region of the apex, not much posteriorly. With great difficulty all of these adhesions were finally freed, but only after it was necessary to traumatize considerably and to displace the heart from time to time. The heart at one time almost stopped, became irregular, and it seemed unwise to progress further. With rest, however, it soon returned to its normal rhythm. Two apex sutures were placed for holding threads. We could then examine the heart well. Both auricles were large; both auricular appendages were engorged with blood and standing stiffly out from the auricular ventricular ring, the left particularly so. They were beating regularly with the ventricles, however. The heart muscle, although it was extensively covered with pericardial adhesions, seemed to be in good shape.

The heart was far larger than we expected. During the manipulation the left hand, without a glove, was on the heart much of the time, and it was interesting to feel the beautiful thrill constantly passing through the organ producing much the same sensation one experiences when feeling a cat that is purring very loudly. In view of the delay necessitated by the separation of pericardial adhesions, we felt it unwise to use the new cardiovalvulotome and, delivering the heart with the apex holding threads, the knife-valvulotome was inserted into the region of the left ventricle about half way from the auriculoventricular ring and directed cautiously toward the mitral orifice. A definite, hard, calcareous ring was encountered, around which the knife was swept in an attempt to calibrate the size of the opening. It was impossible to learn much from this. The knife was placed against what seemed to be the aortic curtain, and a cut was made through something that felt calcareous. The heart was then watched for a definite period of time and the manipulation repeated again through the same opening, with the same sensation of cutting a calcareous tissue. Two sutures were taken to control the slight oozing from the wound in the heart. The heart was replaced in the pericardium, and closure was begun, using continuous silk to the diaphragm and inferior pericardium, continuous silk to the peritoneal opening, interrupted mattress sutures to the linea alba, and continuous silk to the anterior pericardial opening. A double piece of silver wire was then slipped around the sternum; the sternum was held in its correct position and the wire brought taut. Interrupted silk sutures were used to assist in holding the sternum together, while subcutaneous silk stitches and silk were applied to the skin. Closure was made without drainage. Silver foil dressing was used.

The patient was in a fair condition at the end of operation, the blood pressure dropping to around 70 without much pulse pressure. The patient seemed cyanotic, but the heart was regular, and the rate was not excessively rapid. In the next two hours, the condition improved rapidly without the use of any medication except morphin, which was given once for restlessness, and 500 c.c. of a 5 per cent. solution of glucose administered by rectum for warmth and nourishment. It was impossible three or four hours after the operation, to say what had been done or to make definite statements as to the outcome; but at that time, examination revealed no palpable thrill, and the diastolic rumble had disappeared although the loud, snapping, first sound remained. Whether or not, with the vitality of the heart increased, the diastolic rumble would reappear, we could not say. The condition, however, was satisfactory.

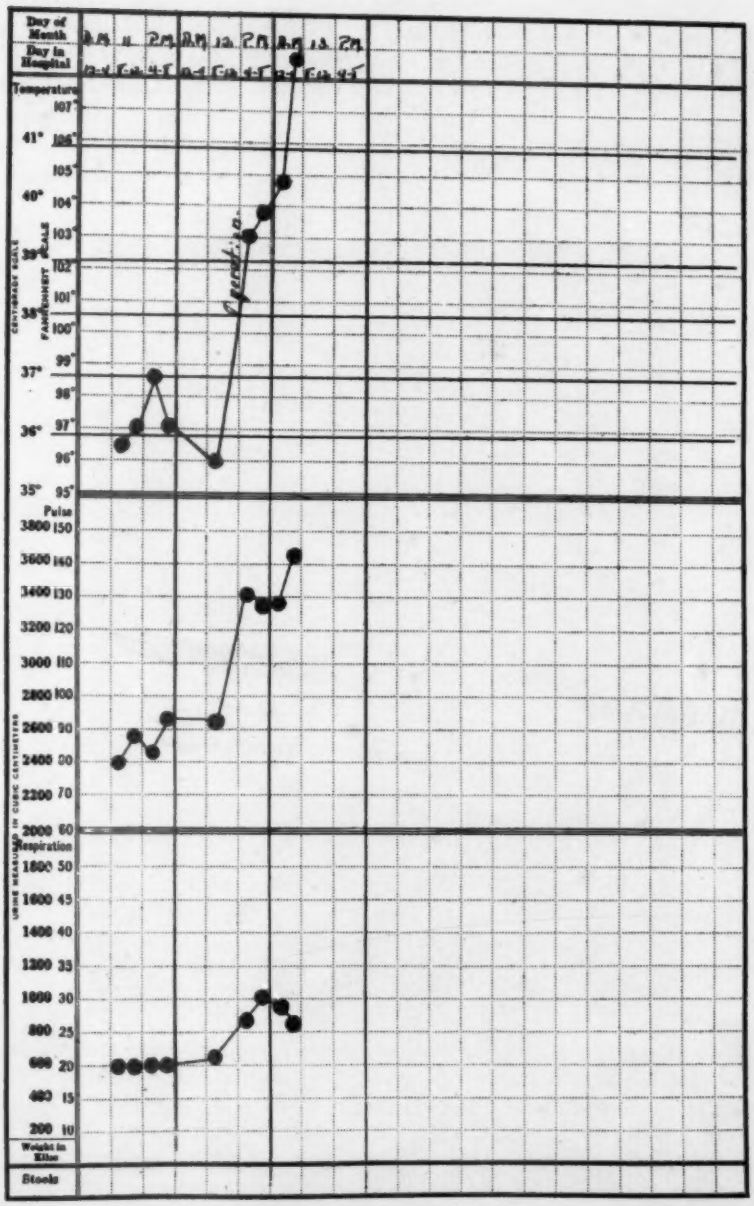


Fig. 45 (Case 3).—Clinical chart.

*Outcome.*—The patient died at about 5:25 a. m., January 13. The general picture following operation was that of a satisfactory recovery followed by a slow but progressive failure, a warning of which was clearly indicated by a rising pulse and a rising temperature (Fig. 45). A few hours after the operation the blood pressure had arisen to: systolic, 95; diastolic, 60. The pulse was 130; respirations, 25. The pressure remained at this point until about 5 p. m.; then it began to fall, but the fall was very slow. It was not until the early hours of the morning that it was reaching a dangerous level. Some morphin was given in the evening. Digitan, 1 c.c., was given intramuscularly

Fig. 46 (Case 3).—Heart, ventricular aspect; pericardial tags and roughness may be noted. Mitral orifice shows divided chordae tendinae and slight notching at either end of long diameter made by knife at operation.

at 10 p. m. Beginning in the early hours of the morning, caffein and epinephrin were given without effect. The patient was constantly under observation, and the only positive finding was that obtained by means of one roentgen-ray picture taken in bed, in which some definite increase in the size of the heart was revealed. There was also some little staining through the dressing. We felt that the heart was probably increasing in size, but we did not feel that it was due to fluid, because we did not expect the fluid to accumulate so rapidly.

Unfortunately, we did not think that the patient's condition warranted the further necessity of taking roentgen-ray pictures. The ultimate picture was one of a failing circulatory apparatus.

*Necropsy.*—January 14, the operative incision was reported. There was a little bloody staining of the fluid about the pericardium. The abdominal incision showed no pathologic changes in the abdomen. The pericardium was distended and full of bloody fluid without clot; there was about 350 c.c. of this fluid. The heart seemed smaller than it was during life. It was removed from

Fig. 47 (Case 3).—Heart, auricular aspect.

the pericardial sac by cutting close to the pericardium. Superficial examination of the heart showed that the sutures in the wounds where the knife had been inserted were holding well. The fluid certainly could not be accounted for by a leakage at these points. The general musculature was in fair condition. It seemed somewhat grayer in appearance than one would have expected, although there was considerable muscle left in the auricular regions, showing that the dilatation of the auricles had probably not been extreme. Both ventricles and auricles showed no evidence of a congenital lesion; the tricuspid valve

was normal, the pulmonic valve was normal, and the aortic valve showed a perfectly competent valve that moved freely, although possibly the orifice on the whole was a little narrowed. There was a beautiful, typical, mitral stenosis with thickening, rumpling, and increased firmness at the border of the opening as well as thickening and shortening of the chordea tendineae. The opening measured 1.3 cm. by 4 mm. At the posterior junction of the cusps the valve had been cut for a distance of only 0.3 cm. At the opposite edge of the mitral ring there was some injury to the musculature, and the largest chorda tendinea had been cut across. Altogether, the orifice was perhaps increased one third in size by these two cuts (Figs. 46 and 47). There were no antemortem or postmortem clots. There were no other findings.

One should deduce from this, as far as surgical intervention is concerned, that a far greater removal of tissue in the ring must be accomplished. From the general point of view, it seems that it may be a mistake to try to close the pericardium anteriorly, at least completely. And possibly, if a small drain had been left in, this patient would have survived and recovered with an improved condition. There certainly was no great hemorrhage, it was probably merely a constant seepage from the division of the many adhesions. All this introduces the interesting question of the proportion of rheumatic hearts that show adherent pericardium. In retrospect, it seems as if death in this case was ultimately due to the rapid accumulation of pericardial fluid. We should have left some opening for the escape of the blood and serum, which was certain to follow the division of so many adhesions, even if the adhesions appeared avascular.

CASE 4.—*History.*—M. M., aged 19, a filing clerk, born in Canada, was admitted to the Peter Bent Brigham Hospital Feb. 27, 1924, with the complaint of shortness of breath and palpitation. The patient's father was living and well. The mother died fourteen years before in labor. One brother was living and well, and two brothers had died in early childhood. There was no family history of cancer, diabetes, gout, tuberculosis, hemophilia, arthritis or diseases of the circulatory, respiratory, digestive, renal, nervous or muscular systems. The patient was self-supporting, rooming and boarding in a girl's rooming house. Her room was on the third floor. The condition of the room was hygienic, and it was well ventilated. The patient needed to work for her own living, and could receive no help from her family; she helped support her father. She was accustomed to get eight or ten hours of uninterrupted sleep, and she worked under no severe tension. There was ample time for relaxation, and she used no tea, coffee, drugs or patent medicines. There was no history of rheumatic fever, chorea, scarlet fever, diphtheria, or pneumonia. The patient had been in the Massachusetts General Hospital one year before for ten days with a fever of unknown origin. At that time she was told that she had heart trouble, and advised to return to the out-patient department. From the female medical department she was sent to the cardiac clinic and had been followed there until her entry in the wards of the hospital in January. There was no history of injuries. Tonsillectomy had been done nine years before but the details of the operation were not remembered.

There had been no headache or trauma. Glasses were not worn, and there was no failing vision, inflammation or pain. The patient had suffered no loss of hearing or any pain or discharge from the ears. She was not subject to head colds, discharge, epistaxis or symptoms of obstruction. The teeth caused no trouble, and there was no history of tonsillitis, sore throat, hoarseness or sore mouth. There had been no pain in the chest, sputum, hemoptysis, or night sweats.

Her appetite was good, and she ate three meals a day regularly. She was constipated, and sometimes used Russian oil. There was no nausea, gas, vomiting, hematemesis, distress, colic, icterus, diarrhea, bloody, tarry or clay colored stools, and no hemorrhoids. There was no nocturia, dysuria, hematuria, pyuria, smoky urine, retention, incontinence, or frequency, and no history of venereal disease. Catamenia had begun when she was 14, and the last period had occurred three weeks before. Periods were regular, and lasted three or four days. No abnormalities of menstruation were present.

The patient was of an even temperament, and had experienced no vertigo, fainting, twitching, spasms, anesthesia, ataxia, girdle, shooting muscular or joint pains. Her memory and disposition were good. She had experienced her greatest weight a year before, when her weight was 103 pounds (46.8 kg.). Her weight on admission was 95 pounds (43 kg.). She had gained one pound (0.45 kg.) in the last three weeks. The patient was an intelligent, cooperative girl who was not unduly mindful of her present condition. She did not react to any extreme degree to factors outside of her life. She did not appear to be of a nervous type and was not introspective; she seemed to be of the suggestive type. Her history could be considered reliable and its retaking in other hospitals warranted its accuracy.

Close questioning failed to reveal any evidence of recurrent tonsillitis, rheumatic fever, or chorea to account for the present illness. The first symptom, that of shortness of breath, dated back to November, 1922, when she found that walking up Beacon Hill by way of Joy Street or the Boston Common steps produced shortness of breath, as well as moderate palpitation; to accomplish this she needed to rest at frequent intervals. This symptom persisted and seemed to progress gradually for the last year until November, 1923, when she noted shortness of breath on such a slight exertion as the taking of a bed bath in the morning. With this symptom she described no orthopnea, edema, or cyanosis.

One morning in November, 1922, she experienced pain in the chest, which was treated with hot mustard poultices, and which was relieved in less than a week. Her illness did not greatly interfere with her work, which was fairly steady and of a light type. She lost two or three days a month from work, and would have absented herself more if she had not been afraid of losing her position. In December, 1923, she was at the cardiac clinic of the Massachusetts General Hospital where she was first informed that her heart condition warranted entry to the hospital. She left her last employment December 31, and entered the medical ward of the Massachusetts General Hospital Jan. 4, 1924, on the advice of her own physician. We visited her there at the request of Dr. P. D. White, who advised her to come to the Peter Bent Brigham Hospital for treatment.

*Physical Examination.*—The patient presented the picture of a well-developed and well-nourished, white girl lying quietly in bed, in no apparent distress, breathing quietly, and mentally clear. Examination of the skull showed it to be symmetrical, without exostoses, irregularities or tenderness. The scalp was clean, and without scars. The hair was black, straight, soft, and of a normal amount. There was some erythema diffusely scattered over the posterior area of the left shoulder. The skin was warm and moist, and its temperature was not elevated. The pupils of the eyes were equal, regular, and reacted to light and accommodation. The sclerae and conjunctivae were not remarkable. There was no photophobia, lacrimation, diplopia, nystagmus, palsies, lid-lag or exophthalmos, and no gross disturbance of the vision. Glasses were

not worn. The ticking of a watch was heard 30 cm. from each ear. There was no discharge, and no stigmas or trophi. There was slight reddening of the meatuses of the nose, but no deformities, obstruction or discharge were present. The breath was bland, and the mouth was without ulcerations, exudate or pigmentation. The color of the lips was normal, and no herpes, ulcerations or fissures were present. The tongue was not coated and protruded in the midline with some tremor. There were no mucous patches. There were numerous capped teeth and much dental work. The color of the gums was normal, and there was no pyorrhea, bleeding or any leadline. The color of the pharynx was normal, and there was no catarrh. The palate was symmetrical, and the gag reflex was present.

The left tonsil was markedly enlarged and inflamed; there was a pale yellow, scattered exudate on its anterior surface. The right tonsil was also slightly enlarged and reddened. The voice was not remarkable. The thyroid gland was not enlarged. There were no bruits, thrills or pulsations, and the lymph glands were not palpable. No stiffness or tracheal tug was present. The thorax was symmetrical, and expansion was equal on the two sides. Its size and shape were not remarkable. The rate, depth and character of the respirations were normal, and there were no abnormal pulsations.

The apex impulse of the heart was in the fifth space, 9 cm. from the midsternal line. Its action was strong and diffuse. The right border of dulness was 4.5 cm; and the left border of dulness, 9.7 cm. from the midsternal line. The upper border was located at the third rib. There was a definitely palpable, diastolic thrill felt all over the precordia, but it was best felt at the apex. The pulmonary shock was easily felt. On auscultation, the rhythm was found to be regular; there was a long, rough, crescendo murmur extending throughout diastole, heard all over the precordia, which was most pronounced at the apex. No systolic murmur was heard at the apex. The sounds were snappy in quality; the pulmonic second sound was greater than the aortic second sound, and accentuated. The first sound at the apex was also snapping in character. There was no aortic diastolic murmur. The regions below the nipples rose equally with deep inspiration. By percussion, the left border was found to come in about 1 cm. on deep inspiration, although the apex impulse and thrill remained palpable at the extreme left. There was no pulsus paradoxus, Broadbent's sign or engorgement of the veins of the neck. The apex impulse did not shift when the patient was in the right, lateral position, but it moved about 1.5 cm. when the patient was on the left side. The supracardiac dulness was 5 cm. in the first space. The radial pulses were somewhat rapid but regular, and the vessel walls were not sclerosed or thickened. The blood pressure was: systolic, 95; diastolic, 55. The percussion note in the lungs was resonant throughout, and the breath sounds were vesicular. The tactile fremitus and whispered voice were normally transmitted, and there were no râles. The lower borders moved normally.

The abdomen was well-developed and symmetrical. There were no pulsations, tenderness, masses or spasm, and resonance was normal. Respiratory movements were not remarkable, and no hernias and no fluid were present. The reflexes were normal. The liver, gallbladder, spleen, kidneys and bladder were not palpable. Rectal and vaginal examinations were not made. The lymphatic glands were not palpable in the neck, axillae, groins or epitrochlear regions. The spine showed no scoliosis, lordosis, rigidity or tenderness. The other bones were not remarkable. There was no involuntary, athetoid or choreiform movements in the arms, and no wasting, tremor, or club fingers. The nails

were normal, as well as the biceps, triceps, and radial periosteal reflexes. There were no joint changes. The legs showed no evidence of varicosities, scars or ulcers, and the knee jerks and Achilles' reflexes were normal. Plantar irritation gave flexor response. There was no Babinski's reflex, Oppenheim's reflex, ankle clonus or Kernig's sign. Test was not made for Romberg's sign, and the patient's gait was not examined. No edema was present, and both feet felt cold to the touch. No joint changes were found. There was no evidence of anesthesia, paresthesia, or hyperesthesia. The ophthalmoscopic examination revealed the fact that the mediums were clear, the disks distinct, and that physiologic cupping was present; the vessels were found to be normal, and there was no exudate or hemorrhage.

*Summary.*—A young woman, Canadian-American, white, aged 19, and single, entered for relief of dyspnea and palpitation. Her past history and family history were unimportant. Her present illness dated back fifteen months, when she had observed shortness of breath on exertion and moderate palpitation. These symptoms had gradually become worse, but were unaccompanied by edema, cyanosis or orthopnea. The physical examination was essentially negative, except for the heart, which showed slight enlargement to the left; a loud, rough diastolic murmur of long duration extending up to a snapping, first heart sound; a palpable precordial thrill, diastolic in time; and a snapping, accentuated second sound. The left tonsil was inflamed.

*Laboratory Findings.*—An examination of the urine, made January 29, showed it to be pale, clear, acid, with a specific gravity of 1.012; albumin and sugar were absent, and the sediment showed no casts, leukocytes or erythrocytes.

Examination of the blood, January 28, revealed: hemoglobin, 95 per cent., red cells, 4,800,000; white cells, 9,600. A stained blood smear showed: polymorphonuclears, 78 per cent.; lymphocytes, 18 per cent.; large mononuclears, 3 per cent., and eosinophils, 1 per cent. The appearance of the cells was normal, and the Wassermann reaction was negative. Examination of the stool, made January 30, showed it to be brown and formed; the benzidin test was negative for occult blood. The result of the phenolsulphonephthalein test, February 8, was 55 per cent. (300 c.c. was voided in two hours and ten minutes). The vital capacity of the lungs, January 29, was 2,200 c.c. or 80 per cent. of the normal capacity; height was 149 cm.; weight was 42 kg.; surface area, 1.35 square meters; the normal, vital capacity of the lungs was 2,700 c.c.

In the roentgen-ray examination, January 29, a 7-foot film of the heart showed it to be just above the limits of normal, in spite of its almost perpendicular position. There was a prominence in the region of the left auricle, compatible with a mitral stenosis. The lungs showed considerable congestion. The measurements of the heart were: Midline to right margin, 4.5; midline to left margin, 6.4; great vessels, 3.7. The internal diameter of the chest was 21.1 cm. The electrocardiographic examination, February 1, revealed right ventricular preponderance.

*Subsequent Preoperative Findings.*—The day after admission to the hospital, the patient complained of a sore throat, and had a fever of 101 F. The tonsils were acutely inflamed, enlarged and showed patches of exudate characteristic of follicular tonsillitis. This quieted down after ordinary, simple remedies,

such as hot gargles and an ice collar, were used. The patient was very little disturbed by this infection, but after three days of normal temperature, the fever returned, and the temperature was elevated as high as 102. The pulse rose to 120, and the respirations to 30. There were no signs found on physical examination except some moist râles at the bases of both lungs. The white count rose to 16,000 and then to 22,000. Blood cultures were negative. During the height of this infection, the urine showed a trace of albumin, some leukocytes and occasional granular and hyaline casts. The general examination of the patient remained essentially unchanged.

The vital capacity of the lungs fell somewhat with the infection, but stayed in the vicinity of 70 per cent. normal. The blood pressure varied between 85 and 100 systolic, and 50 and 65 diastolic.

The patient was seen by a number of observers, and all agreed that she had marked constriction of the mitral valve but no evidence of congestive failure. There was a slight question as to whether or not in addition she had any pericardial adhesions. She was well compensated and after the infection, felt very well and remained free from fever for two weeks. She was dependent on her own work for support, but realized that even a slight effort brought on the shortness of breath. Our studies indicated that the mechanical factor was the important one, and that the heart musculature was healthy. The social status of this patient prevented her from living a sedentary life. An operation was proposed to her as offering some possibility of improving her health, although the dangers attending it were also made clear. This decision was accepted by her, and the father also gave permission for the operation.

*Operation.*—Feb. 25, 1924, partial valvulectomy for mitral stenosis was performed (Fig. 48), under ether (Connell apparatus). The anesthesia was well taken, and the procedure was carried out smoothly and with great satisfaction throughout the operation. Preoperative medication was administered in the form of morphin, 10 mg.; atropin, 1 mg. The usual alcohol and bichlorid preparation of the chest was made. The position of the patient was semirecumbent. A midline incision was made from the tip of the manubrium to within one inch of the umbilicus, carried through to the sternum and linea alba below the ensiform. The linea alba was divided to the peritoneum, and a finger was thrust under the ensiform and up beneath the sternum to dissect the tissues away from it. A spatula was laid beneath the sternum, by means of the circular, motor driven saw, the sternum was cut in the midline up to the second interspace, and cut across at this point. The two halves of the sternum were pulled out with retractors; the pleura was wiped away on both sides, a small hole being made in the left pleura, which was closed with a stitch after a little air had entered the pleural cavity. The peritoneum was then opened over the liver; the diaphragm and adherent pericardium were inferiorly slit up to the crus; the anterior pericardium was opened up towards the arch of the pulmonary artery, and the two halves of the sternum with attached tissues held apart by the rib-spreader retractor.

There was about 100 c.c. of free, light colored fluid in the pericardial cavity, and no adhesions. The muscle was covered with a rather gray film but seemed to be in a good condition and well-nourished. Auricles and ventricles were beating regularly; there was no fibrillation; the left auricle was considerably enlarged, but the enlargement was mostly posterior. It was far more tense than the right, and the left auricular appendage was so tense that it felt almost as if it contained an organized thrombus. The apex finger was pushed down

into the auricle until an opening was felt at the mitral region, but it would not admit the whole of the index finger. An apex suture was placed in the heart. Palpation of the heart revealed the fact that one could feel a very definite thrill over the left ventricle, but that one could not feel any with the hand on the right ventricle. Pulling the apex threads mesially and upwards gave a good exposure of the left border of the left ventricle. Deep silk sutures were then placed approximately 1 cm. apart about an inch and a half (3.77 cm.) from the apex. An incision with the knife was made into the ventricle between these sutures; the sutures were crossed and the cardioval-

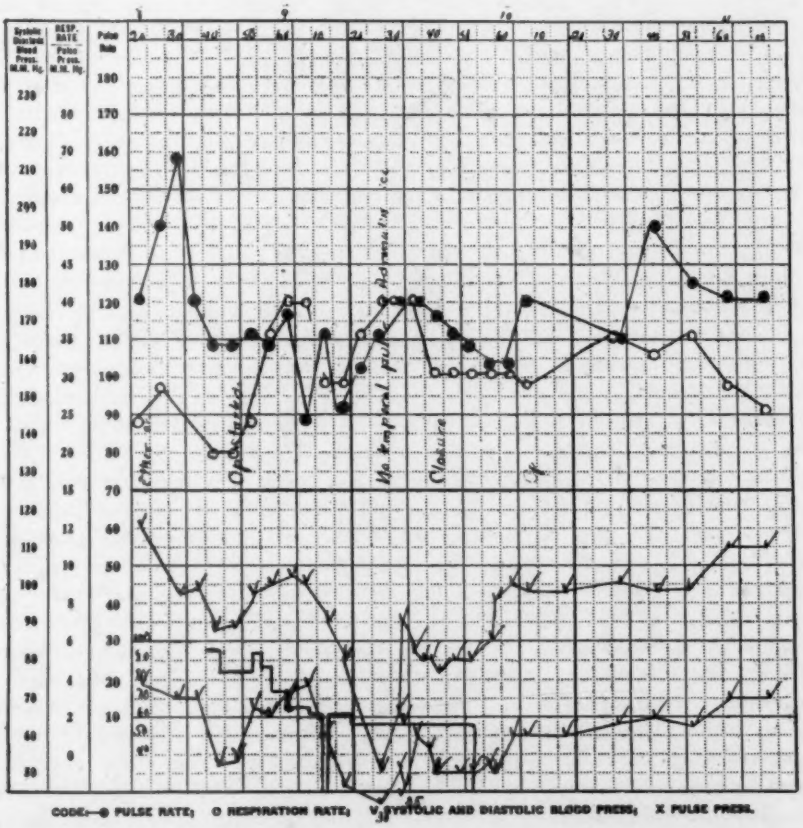


Fig. 48 (Case 4).—Record of pulse and respiratory rates, blood pressure and ether vapor tension throughout operation.

vulotome (fourth modification), was then inserted in the opening in the ventricle and pushed into the ventricle, while the tip of the left forefinger invaginated the auricular wall towards the mitral orifice. With the instrument pushing towards this finger, the orifice was felt for and the tip of the instrument pushed up into the left auricle. The instrument was then allowed to open and, as the instrument was withdrawn, it was felt to lie in a fixed position, which we took as evidence of its being at the valve edge. It was then forced mesially, and a very dense, hard structure was encountered, which was cut with a snapping

noise. The instrument was withdrawn with a rather large piece of tissue attached to it. Three interrupted sutures were used to close the opening in the ventricle; the ventricles maintained a regular rate throughout. The pericardium was wiped free of a small amount of blood and left filled with salt solution. The inferior portion of pericardium and diaphragm were closed with a running suture of C silk, and the peritoneum was closed with a suture of the same material; the anterior pericardium was closed with a suture of the same material down to its lowest point, where an opening of about an inch (2.5 cm.) in length was left in the pericardium in order to make allowance for the escape of fluid into the mediastinum in case too much should accumulate. This was done because of our experience in Case 3. Two silver wire sutures were placed about the sternum. Closure of the linea alba was made with interrupted sutures of silk, silk to the subcutaneous tissues and skin, and silver foil dressing. The patient made a good, immediate recovery. An hour after the operation the blood pressure was the same as it had been before the operation. The patient was conscious, and the pulse rate was regular, full, satisfactory. Because of the fact that some air was allowed to enter the left chest cavity, the sounds were somewhat muffled after the operation, but they certainly were changed in quality. The piece of tissue removed was about 1.5 by 1 cm. and consisted of fibrous tissue and what seemed to be a firm, calcareous center and the attached edges of the chorda tendinea.

*Outcome.*—February 27, the patient was making a good, immediate, postoperative recovery. Within two hours after the operation she was conscious and in good condition. The blood pressure was around 95, and the pulse, after the operation, was approximately 120, where it remained for some hours and then rose steadily to about 140; it was regular and came through well at the wrist. The patient developed a postoperative temperaure (102 F. plus), which had been maintained up to the present time. The respiratory rate had steadily increased until it was then around 46. The blood pressure had fluctuated above and below 90, sometimes touching 100 and sometimes as low as 86; it was never lower, and there was a fair pulse pressure. Repeated roentgen-ray studies showed a gradual, slight increase in the size of the heart. We were dubious as to whether this was due to dilatation or to the presence of fluid in the pericardial sac. At the left, lower back there were all the signs of either consolidation or collapse, with bronchial breathing and râles, due to pressure of the enlarged heart. The patient seemed to have postoperative pneumonia. The white cell count was 23,000. This, however, should be taken as a good omen rather than otherwise, for patients who can react hold more promise than those who cannot. The heart sounds were regular and rather distant; this was complicated by two factors, one, the presence of a small amount of air in the left pleural cavity, and two, the possibility that the heart was surrounded by fluid. There was unquestionably a great change in the murmur. The thrill was not felt, and the diastolic rumble seemed to be absent. Almost no medication had been used except small amounts of morphin, until that morning, when ouabin (0.00025 gm.) was given in the right basilic vein. This seemed to produce no change. Hot water bottles were applied; digitalis folia was given once, but it promptly induced vomiting and seemed to cause considerable distress. Fluid intake had been maintained fairly high, about 1,700 c.c. during each of the first twenty-four hours. The patient did not appear desiccated, although unquestionably it would have been better if she could have obtained more fluid. There was some complaint of pain in the

incision, but not nearly as much complaint as in previous cases, which would seem to point out that the saw creates a more satisfactory wound in the sternum than the bone-cutting forceps. Today, a loud, harsh, to-and-fro, pericardial friction rub was heard. The abdomen was normal, and the patient voided spontaneously. Her condition was critical but hopeful.

On February 28, the report was that by 8 p. m. of the night of February 27 the patient seemed distinctly improved. The pressure, systolic, had risen to 100; the pulse dropped to 120, the temperature to 100 and respirations to 35, and we retired for the night feeling that she had conquered the postoperative pulmonary complication and that from now on the prognosis would be improved. This, however, was premature. By 4 a. m. that morning, the pressure had dropped to about 85, and the pulse was very thready. The patient was cyanotic. Previous to this, 500 c.c. of salt solution had been given under the right breast; the patient had taken one glass of ginger ale and, with 6 mg. morphin administered twice, she had had a fairly comfortable night. Ouabin 0.0003 gm. was given intravenously, at the time when the patient's pulse had disappeared and no pressure could be obtained. This was immediately after a roentgen-ray picture had been taken in bed, the effort having considerably disconcerted the patient; we felt the effort necessary for the information to be derived. After taking the ouabain, the patient steadily improved; the pulse had dropped in rate and pressure had returned to around 85. The roentgenogram showed an appreciable increase in the heart shadow. In spite of this, with the facts staring us in the face that the patient must be going under from heart failure, probably more pronounced in the left side of the heart than in the right, the possibility of tamponade from a pericardial effusion could not be ruled out. A needle was therefore inserted in the fifth interspace, nipple-line, dry tap. It was felt unwise to open the old wound, as we felt that the patient would not be able to stand the manipulation. There seemed to be nothing to do but to keep the patient warm and comfortable, and to give her oxygen. After temporary benefit from the use of ouabain, the pulse began gradually to disappear again, and at 9 a. m. 0.5 gm. caffein was given subcutaneously. Following this, there was a slow but continous improvement up to 11 a. m. The blood pressure at that time was 100, and there was still some hope.

According to the report of March 1, the pressure had remained the day before at 100 or over; respirations were around 35, and the pulse around 125. The patient appeared lethargic, and from time to time there were periods of deep cyanosis. She had great difficulty in raising tenacious sputum; the effect of getting it up exhausted her. The heart showed a broad, pericardial arch. There was definitely bilateral pneumonia. The white count continued to be elevated (22,000 the day before), and the temperature remained above 103 F. most of the time. It seemed to be entirely a question of whether or not the patient could shake off the pulmonary infection. Late in the evening, the pressure had risen as high as 110. On the morning of March 1, at 8:00 a. m., the pressure dropped again, though only to 90; 0.5 gm. caffein at once restored the pressure to the previous level of around 105 systolic. There seemed to be a slow, continuous rise in the diastolic pressure, which of course was a bad sign. There was also a peculiar sweet odor to the breath similar to acetone, but a specimen of urine showed no acid bodies that morning. With the lapse of so much time, it seemed as if she might be able to conquer the infection, in spite of the fact that her condition March 1 was almost as critical as it had been two days before.

The patient died at 7:30 a. m., March 2. The end was not unexpected in view of the long and stormy pneumonia convalescence, although each succeeding day, with the patient's condition still maintaining a fair level, gave hope that possibly she could overcome the process in her lungs (Fig. 49). Necropsy proved that, instead of this happening, the progress of consolidation had been steadily increasing. Twelve hours before death, the white blood count was 32,000 and the temperature 102 F.; the pulse remained in the last twenty-four hours around 120 or 130, and respirations gradually rose from 30 to 30 plus. The blood pressure remained at a good level the day preceding death, always over 100, although 0.5 gm. of caffein was given once for a sudden, unexplained drop. It was necessary to give morphin in small doses for restlessness, and the intake of fluid was maintained at a good level. No acetone bodies appeared in the urine, although her breath was sweet and of the odor of acetone. She was seen at 6 a. m. the day of death, and from that time on it was obvious that the pressure was rapidly dropping and that the pneumonia process had so increased that there was no hope of recovery. She became very restless, the respirations rapidly rose, blood pressure fell to nothing and, in spite of caffein, the end came quite quickly.

The outcome in this case was a great disappointment because in this case the patient was an apparently good risk. The heart muscle was in good shape, and there was a mechanical stenosis. There was no evidence of anything complicating the picture except possibly the fact that about two weeks before operation there was an unexplained fever which, however, we did not believe to be of intracardial origin. Studies made in the very trying week after operation would seem to indicate that there was not any accumulation of fluid in the pericardial sac, and that therefore death could not be placed at the doors of heart tamponade. The maintenance of the high blood pressure and vigorous heart action throughout this very stormy period proved that the heart itself was doing as well as could be expected, and that failure could not be placed at the doors of the myocardium. We had indication from the begining that there was extensive pulmonary involvement. It is true, usually, that postoperative pneumonia is of a transient nature and not of this type. Possibly, the fact that some air entered the left pleural cavity mitigated against our chances in the face of pneumonia. This may be particularly true since, in the light of some recent publications on postoperative lung complications, it has been stated that small areas of atelectases occurring after operation are often the seat of pneumonic processes.

Directly after the operation, the prolonged diastolic thrill which had been present before the operation became imperceptible. The heart action was regular, the first sound not accentuated, and there was a questionable, presystolic murmur between the nipple and the left, lower sternum. Frequent auscultation during the succeeding seven days revealed a marked change in the sounds of the precordium. All that was left of the diastolic rumbling, that could be heard anywhere over the precordium, was a very short presystolic murmur. A slight systolic blow was present at the apex at all times; no true thrill could be made out, although sometimes a short vibration, which was difficult to time, was felt near the left nipple. On February 27, a definite to-and-fro pericardial friction was first heard over the base of the heart. The following day, this friction rub became more extensive and gradually became audible all over the precordium. At the left, lower back, bronchial breathing, slight dulness, and increased whispering was distinguished, and there were some râles at the right base. The heart dimensions, as indicated by the roentgen-ray examination,

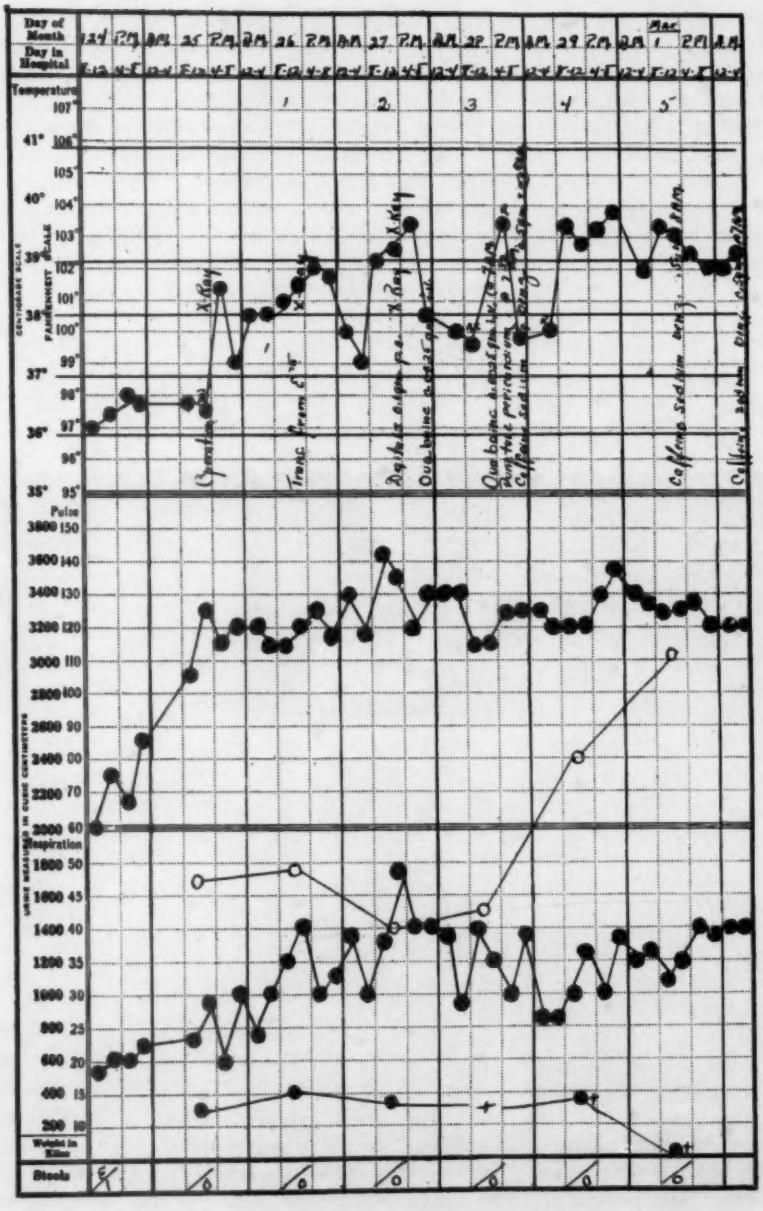


Fig. 49 (Case 4).—Clinical chart, showing postoperative course.

slowly increased, although not to any large extent (Fig. 50). It seemed as if there might be a moderate pericardial effusion, and for this reason the pericardium was tapped in the left fifth interspace. No fluid was obtained. The heart was definitely felt with the tip of the needle, and it was certain that we were in the pericardial cavity. This was done February 28. A roentgenogram showed the presence of air in the left upper chest; there was, on physical examination, hyperresonance and diminished breath sounds in this area.

During this postoperative period, the patient was combating three conditions; namely, a left pneumothorax, an acute pericarditis, and, what was by far the most important, pnuemonia, involving both chests. The patient's appearance was constantly one of pallor with slight cyanosis; the pulse remained small, at various times disappearing entirely, although it responded to ouabain or caffein. The patient seemed to be physically and mentally exhausted, complained very little, and was constantly trying to get some mucous out of her throat. At first, it was thought that the increased aperture of the mitral valve made at the operation might have been too much for the left ventricle. After necropsy, we doubted very much if this had anything to do with the patient's death. I think the essential complication was infection of the lungs and the subsequent toxemia.

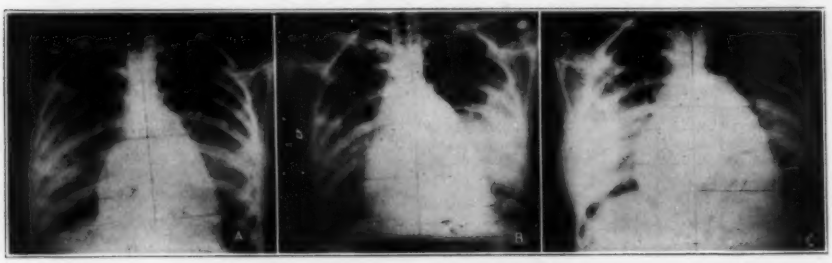

Fig. 50 (Case 4).—Roentgen-ray studies of heart; *A* represents preoperative plate; *B*, plate twenty-four hours postoperative, and *C*, plate five days postoperative. The partial left pneumothorax is seen in *B* and *C*; the heart shadow is slightly increased in size. The pulmonary field shows increasing density.

*Necropsy.*—A restricted necropsy occurred March 2, at 3 p. m., eight hours after death. The case was diagnosed as chronic fibrous endocarditis with mitral stenosis, with an operative wound of the heart, pericardium and chest wall, acute pericarditis, edema of the lungs, chronic, passive congestion of the lungs, and bilateral bronchopneumonia.

The body was that of a well-developed and well-nourished, young adult, measuring 150 cm. in length. Rigor mortis was complete. Postmortem lividity was present in the dependent portions of the body. The scalp was covered with an abundance of long, black hair which appeared normal. The ears and nose were normal to external examination. The pupils were round and equal, and the sclerae were white. The conjunctivae were smooth and glistening. The lips were blue in color. The superficial lymph nodes in the cervical and axillary regions were small; the inguinal lymph nodes were small. The chest was symmetrical, and the breasts contained no palpable masses. The abdomen was rounded, but not distended. The back was normal, and the extremities showed no evidence of edema. The skin over the entire body was smooth and white, normal and elastic.

The operative wound was reopened. In the fat just beneath the skin, at a point opposite the insertion of about the sixth costal cartilage, there was a small pocket of grumous, purulent material similar to liquefied fat plus a few white cells, possibly 1 c.c. in all. This seemed to be quite well walled off and to have no connection with the skin above or the tissues beneath the sternum below. Smears from this showed what looked like staphylococci. The rest of the wound was clean and well healed. The silver wire sutures surrounding the sternum were then cut and the sternum retracted, with the escape at once of some straw-colored fluid; as one looked in one could see that the opening left in the pericardiac sac was still patent, and that this fluid connected with the fluid in the pericardial sac. All of this fluid was carefully collected, and it did not measure more than 150 c.c. at the very most, if indeed there was that much. It was a straw-colored fluid and did not appear to contain very many cells. The sternum was then quite widely retracted, the pericardial sac opened and one could see that the heart itself was plastered over with a delicate, fibrinous, jelly-like, thin covering which was adherent in places to the pericardium, in other places to the heart. It was exactly the condition that we had often seen in dogs and which we think often occurs when there is no infection, presumably due to trauma or hot salt. The wound in the heart was perfectly healed, and there was not a sign of blood in the pericardial cavity. The heart was not dilated, but this fibrinogelatinous exudate had glued the auricular appendages to the myocardium below and had filled up to the crevices about the auriculoventricular ring. The lung was obviously greatly solidified, and there was more air in the left pleural cavity than in the right when the pleura was opened, the upper lobe being somewhat depressed and collapsed on that side. In order to facilitate examination, the heart, pericardium and lungs were removed in toto. When they were removed it was seen that the lungs were everywhere solidified; they stood out as voluminous, heavy organs. When they were separated from the heart their weight was found to be 1,300 gm., and a frothy, edematous fluid came from the bronchi as the lungs were squeezed; they were wet lungs. There was no fluid in the pleural cavity, and no evidence of acute pleurisy. The heart was not weighed. The left auricular appendage was then opened, and one looked in at the mitral valve. It was extremely interesting, and showed how well the new instrument had performed its task. It was one of those narrowed, oval, slit-like, valve openings; the instrument had broken through the aortic leaflet, so that there were two openings. The instrument had been thrust through the cusp and then had come back, cutting out a large piece, giving a very considerable opening between the auricle and ventricle in the aortic curtain close to the edge of the stenosed orifice. The heart was then washed free of clots, closed and injected with formaldehyd; formaldehyd cannulas were left in both auricles and the heart in a jar of formaldehyd overnight, so that the heart would be fixed in the distended position.

The peritoneum was smooth and glistening. There was no free fluid, and there were no adhesions. The liver and spleen were normally situated and appeared to be normal in size and shape. The surfaces were both smooth and glistening. The estimated weight of the spleen was 150 gm. The liver probably weighed in the neighborhood of 1,500 gm. The gallbladder was moderately distended. It contained no palpable stones. The foramen of Winslow was open, and the lesser peritoneal cavity was free. The pancreas was normal; on external examination, palpation of it revealed no abnormalities. The

mesenteric lymph nodes were small. The organs of the pelvis were free from adhesions. The uterus was normal in size, shape and position. The fallopian tubes and ovaries were negative.

The weight of the lungs was 1,300 gm. Both lungs were voluminous. They did not collapse when the pleural cavities were opened, nor did they collapse when they were removed from the thorax. The surfaces were smooth, glistening and moist. There was a moderate amount of carbon pigment visible through the pleura. Surfaces were mottled throughout, with small, irregular areas of dull red interspersed with gray lung tissue. The lower and posterior portions of the inferior lobes were deep red, and on the surface of these lobes there was a very thin layer of fibrinous exudate. These portions of the lungs

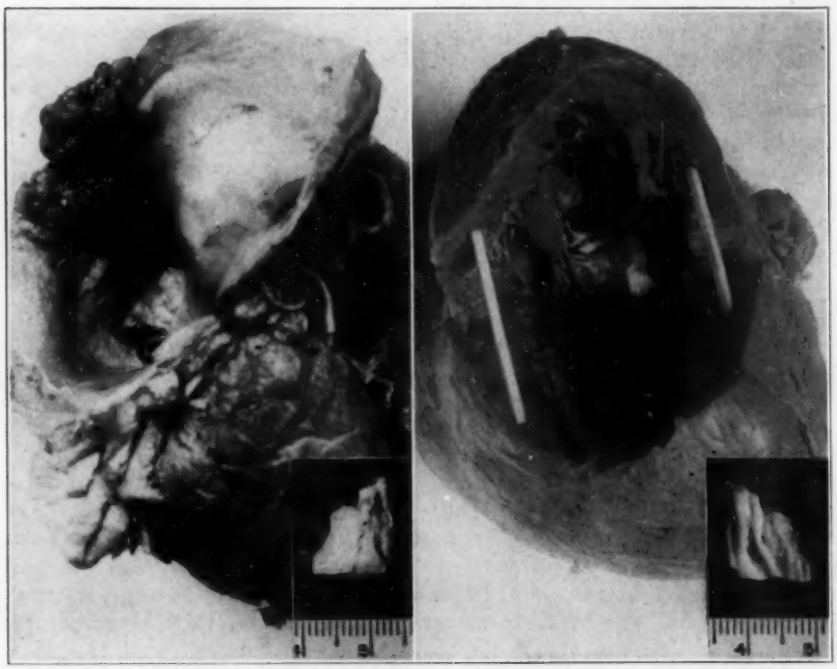

Fig. 51 (Case 4).—Auricular and ventricular views of mitral orifice; insets represent the auricular and ventricular views of valve segment removed at operation. The operative defect occurs in the aortic cusp without involving the margin of the stenosed valve.

were uniformly firm in consistency. Palpation of other areas revealed patchy areas of increased consistency. Frothy fluid was expressed from the trachea with slight pressure. The lungs were to be fixed in Kaiserling's solution before sectioning.

According to the bacteriologic report, cultures made from the surgical incision and pericardial fluid showed the presence of *Staphylococcus albus.*

*Microscopic Examination.*—Five sections were cut from representative areas in each of the five lobes of the lungs. In general, the microscopic pictures presented in these sections were quite alike. The outstanding features in each

instance were marked chronic passive congestion, marked edema, and an inflammatory reaction confined principally to the alveolar walls and the connective tissue septums. Evidence of chronic passive congestion was found in the presence of relatively large numbers of large mononuclear phagocytes in the alveoli, many of which contained a considerable amount of golden brown hemosiderin pigment. Occasionally, some of the alveolar walls appeared to possess an excess of connective tissue. This condition was not pronounced,

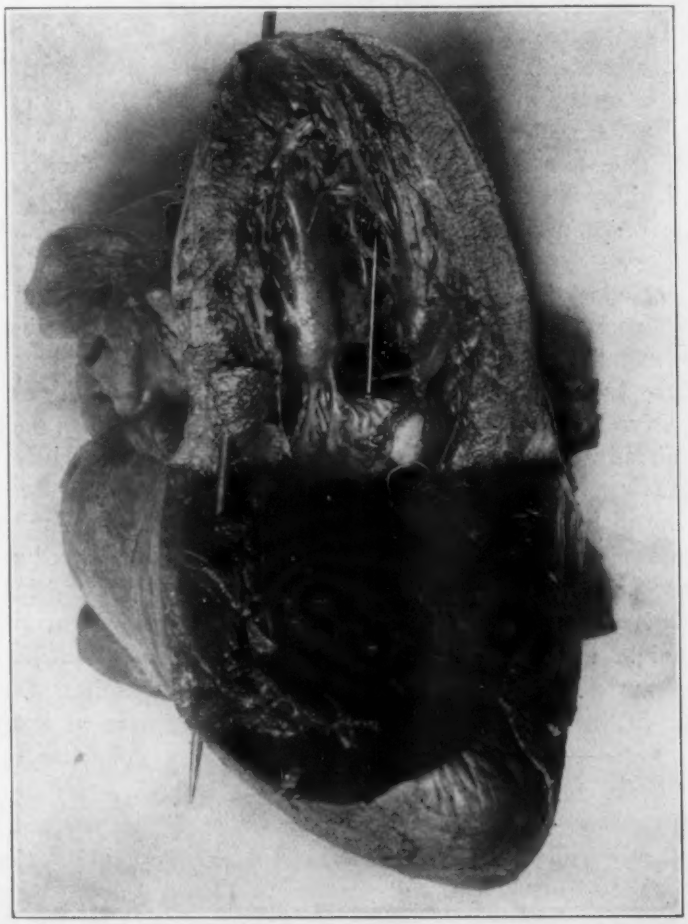

Fig. 52 (Case 4).—Ventricular view; the point of the pin rests against the endocardium at the site of the incision. No thrombus was present.

and was found only in certain small areas. In addition to the large mononuclears in the alveoli, there was an abundance of a fine, granular, eosin staining precipitate. This finding confirmed the gross diagnosis of marked edema. The alveolar walls were quite generally thickened. The increase in thickness in most places was apparently due to the presence of an acute inflammatory exudate, rich in polynuclears. There were focal areas where the alveoli were full of red blood corpuscles, but this was infrequently seen, and the bronchi were entirely free from cellular exudate.

The fact that both lungs were voluminous, and of slightly increased consistency is probably explained principally by the marked edema. Other factors tending to produce this gross appearance were the increase in thickness of the alveolar walls and the presence of large mononuclear phagocytes in the walls.

The heart was opened later and photographs taken (Figs. 51 and 52).

### COMMENT ON CASES

From such a limited experience no final deduction can be drawn either for or against the proposal or the procedure. We feel, however, that much has been learned that should be of value in the consideration of such cases and in subsequent operations. Certainly, there can be no doubt that the method of exposure used is satisfactory, simple in execution and that it apparently produced no especially harmful effect on our patients. The fact that there were no operative deaths, or any indication that the procedure per se was a factor in the subsequent fatalities is comforting.

A mortality of 75 per cent. is alarming, but to those who will analyze the full reports of the separate cases it may not appear so disastrous. The three fatalities seem fully explained by causes not inherent in the procedure itself, except in Case 3, in which the slight but continuous oozing from the divided cardiopericardial adhesions added to the burden of an already diseased heart. Death in Case 2 was obviously due to cardiac failure. We might in retrospect think that this type of case, in which fibrillation gave evidence of considerably advanced myocardial damage, was too poor a risk to be considered for such a procedure. But even our judgment here may be faulty because, if we could have produced in such a case a real decrease in the mechanical obstruction, great relief might have occurred. Of course we do not know how the diseased auricle would have borne the increased regurgitation, or how the damaged ventricle would have tolerated its increased amount of blood. In Case 4, a common, and often serious, pulmonary complication [102] was apparently a factor in the fatal issue.

Indeed there are so many questions, obviously unanswered before the first and even after the last operation, that we feel that our mortality rate should be judged, if one wishes to use this as a criterion, only in comparison with figures obtained in the early surgery of other parts of the body, when similar important questions were still unanswered. May we recall the mortality figures in the early surgery of such a relatively simple field as that of the stomach, collected by Dr. W. W. Keen [103] for his Cartwright lectures. Of the first twenty-eight gastrostomies, collected in 1875, all the patients died, and in a series of thirty-five gastroenterostomies in 1885 the operative mortality was 65.7 per cent. Moreover, it took years for these figures to improve. In 1884, the mortality for gastrostomy was still 81.6 per cent.

We hope that the experience gained from the attempts made in each case has been put to good use. The failure to abolish the thrill and diastolic murmur in Case 1, and the opportunity to see in Cases 2 and 3 how little could be accomplished with a small knife, confirmed our doubts regarding the possible efficacy of such an instrument to accomplish this task; this led us to further investigations resulting in the development of the powerful device (cardiovalvulotome) used in the last operation. The fact that this instrument will accomplish the desired result was proved by the examination of the heart in this case. We hope that further opportunity to use this instrument will arise.

This is not the place to consider in detail adhesive pericarditis. The fact that cardiopericardial adhesions occur, and occur frequently, in rheumatic heart disease is well known. There are no absolutely reliable signs or symptoms by which it can be readily diagnosed, but our experience in Case 3 is of value and interest because it should impress any one interested in similar attempts with the possibility of encountering a similar condition. In such a circumstance, the correct procedure would unquestionably be to do nothing beyond a simple reduction of the bony wall, or some modification of pericardiectomy and cardiac decompression suited to the special indication in the particular case. To go ahead with the valve operation in addition to such an ordeal is certainly contraindicated and we fully recognize our error in this regard in Case 3.

The possibility of a postoperative accumulation of fluid in the pericardium, due to mechanical irritation and trauma, has made us consider repeatedly whether or not drainage should be used in these cases. In Case 1 there was such an accumulation; in Case 3 the patient died of "heart-tamponade," and we were so impressed by this factor that in Case 4 we left a small opening in the pericardium, hoping that if an effusion should form it might drain out and be absorbed in the mediastium. However, there is no doubt but that drainage [104] will encourage adhesions; thus, in time it will tend to establish a condition quite as serious perhaps as the original one. It might be wise to establish drainage only in cases in which, because of the division of cardiopericardial adhesions, postoperative effusion is almost certain to occur.

It is possibly of interest to refer to the status of auricular fibrillation in Case 2, as this condition in man has never before been examined by the eye. There was no question as to the diagnosis; electrocardiographic studies had been made at least one and one-half years previously when the condition was first noted, and these were verified by similar studies at this time. With the heart exposed, it was noted that both auricles consisted of huge, dark bags of blood resembling in color and in their apparent consistency any large venous structure. No contraction or intrinsic movement was noted by any one, except a rare quiver in the right auricular appendix. The findings in Cases 3 and 4, in relation to

the origin of the thrill in mitral stenosis, were also made in man for the first time. When the hand was placed on the left ventricle, there was experienced exactly the same sensation felt when one's hand is placed on a cat that is purring loudly. This sensation could not be obtained over either the right auricle or the right ventricle, and there was but a suggestion of it over the left auricle. The thrill of mitral stenosis is produced in the left ventricle.

The tolerance that such cases had for ether anesthesia, the ability of the heart to withstand manipulation, the effect of hot salt and epinephrin applied externally when the cardiac contractions were excited and the many little improvements in the technical performance of the operation are well exemplified in the protocols and need no elaboration here.

We feel that the proposal that certain cases of mitral stenosis may be relieved by surgery has not been contradicted by our experiences, and we hope that similar opportunities in other cases will prove that the proposition is well founded and desirable.

## X. SUMMARY

It is our opinion that there are cases of mitral stenosis in which the mechanical obstruction plays the dominant rôle. The diagnosis of this affection is made on the physical findings, and particularly on the palpation of a diastolic thrill at the apex; on the existence of a characteristic diastolic murmur (except in some cases of fibrillation); on the indirect electrocardiographic evidence of right ventricular preponderance and changes in the auricular complex (P wave), and on the roentgenologic findings of an increased prominence in the region of the left auricle. On the basis of the importance of the factor of mechanical obstruction, and because of the experimental evidence of the improvement in blood pressures within the chambers of the heart with the reduction of an artificially created stenosis, operative intervention in such cases seems indicated.

Experimental data are presented corroborating the results obtained by previous investigators that in animals sectioning of the mitral valve can be performed by various methods with a high degree of assurance that each attempt will be successful and without great risk to the animal. Several of such methods as well as the development of a new instrument capable of cutting a fibrosed and even calcareous mitral orifice are described in full with the protocols of illustrative experiments.

In a discussion of the various operative methods reasons are given favoring the apex of the left ventricle as the place of choice for the insertion of the instrument to be used in reducing the stenosis. Moreover, an instrument well suited to this task is the cardiovalvulotome described in this communication.

The operative technic for performing partial valvulectomy in man for mitral stenosis is described.

Four cases of mitral stenosis, in which an attempt was made to relieve the obstruction, are cited in full. The first patient is living and apparently improved one year after the operation. The other three cases survived the operation ten hours, twenty hours, and six days respectively. In one of these latter cases, an undiagnosed adherent pericardium complicated the picture and was a direct factor in the fatality. In the last case, death was due to bilateral, postoperative bronchopneumonia. The cardiovalvulotome was used only in the last case, when it actually excised a segment from the stenosed valve and made possible a greater flow of blood from auricle to ventricle.

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101a. Sept. 1, 1924, the condition was further improved. There was a gain of 9½ pounds since operation. The patient was at a summer camp, canoeing and in wading daily. She had no dyspnea, no precordial pain, and no hemoptysis.

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# EXPERIMENTAL STUDIES IN ARTERIOVENOUS FISTULAS *

## I. BLOOD VOLUME VARIATIONS

EMILE HOLMAN, M.D.
Resident Surgeon, Peter Bent Brigham Hospital
BOSTON

In 1890, Harris H. Branham [1] of Brunswick, Georgia, described an aneurysmal varix of the femoral vessels following a gunshot wound, and concluded his article with this comment:

> The most mysterious phenomenon connected with the case, one which I have not been able to explain myself or to obtain a satisfactory reason for from others, was slowing of the heart's beat when compression of the common femoral was employed. . . . This began to be noticeable after the wound had entirely healed and became more marked until pressure of the artery above the wound caused the heart's beat to fall from 80 to 35 or 40 per minute, and so to remain until the pressure was relieved. Compression of the artery of the sound limb produced no such effect. Attending the slowing of the heart was a slight dizziness and some dyspnea. Examination of the heart showed it to be free from valvular trouble.
>
> I report this case because of its unique character in that the varix was caused by the passage of the ball between the vein and artery and also to elicit some information as to the correct cause of the retarded heart action.

Incidentally, it may be noted that this intrepid surgeon cured his patient by ligating the artery above and below the fistula with silk, the wound healing by first intention.

In 1924, thirty-four years later, Gessner [2] reported a case of aneurysmal varix from the Matas Clinic in New Orleans, with this comment:

> A curious observation was that of blood pressure. The pressure just above the patella was 120 systolic on the normal side, only 80 on the affected side. This was to be expected on account of the diversion of a part of the arterial blood into the vein. But a *strange* and *inexplicable* observation made in this connection was that while the pressure at the groin on the normal side was 140/70, that just above the swelling on the affected side ranged from a systolic reading of 240 mm. down to 0, the arterial beat being heard when no pressure was recorded by the manometer.

As it will be demonstrated later, both the "mysterious phenomenon" of Branham's case, and the "strange and inexplicable observation" in

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* From the Laboratory of Surgical Research, Harvard Medical School, and the Hunterian Laboratory of Experimental Surgery, Johns Hopkins Medical School.

1. Branham, Harris H.: Internat. J. Surg. **2**:250 (Nov.) 1890.
2. Gessner: Ann. Surg. **79**:370-374 (March) 1924.

the Matas Clinic case are dependent on the same physiologic factors. In the July issue of THE ARCHIVES OF SURGERY, 1923,[3] a case was presented which threw light on the question by providing the observation that the changes in pulse rate were accompanied by remarkable coincident changes in general blood pressure. A popliteal arteriovenous fistula of twenty-five years' duration exhibited a markedly enlarged heart with a large dilated artery leading to the fistula, as disclosed at operation (Fig. 1). Closure of the fistula by simple compression caused a momentary increase in general blood pressure from 118/66 to 160/90, with a rapid subsidence to 126/88 where it remained until the fistula was again opened; then it fell precipitately to 96/48, with rapid recovery to 116/66 (Fig. 2). Pulse variations accompanied these blood pressure changes, a pulse of 76 dropping to 38 when the fistula was closed, with recovery to 80 when the fistula was opened.

These observations were so striking that they arrested one's attention, until an explanation of both these phenomena was developed by consideration of the whole problem from the point of view of the physiology of the circulation of blood. Reduced to its simplest terms, and with particular reference to blood pressure, the general circulatory system consists of: (1) a central pump, the heart, as a source of propulsive power; (2) a network of vascular tubes, the efferent arteries and the afferent veins, with (3) an intermediate capillary bed providing a more or less constant peripheral resistance. The variable factors in such a vital system are: (1) the rate and force with which the pump acts, or its output per unit of time; (2) the total volume of circulating fluid; (3) the total capacity of the system as influenced by contraction or dilatation of the vascular bed, and (4) the peripheral resistance, as represented by the capillary bed.

By altering any one of these factors one would expect an immediate effect reflected in the general blood pressure, provided compensatory changes were not too rapid to prevent detection of these fluctuations in pressure. It is well known to all physiologists that tremendous, immediate fluctuations in general blood pressure can be produced by exsanguinating an animal, by injecting vasodilator or vasoconstrictor drugs, and even by so-called hydremic plethora. The introduction of an arteriovenous fistula into the system permits observations as to the effect of a permanent alteration in one of the variable factors, namely a decrease in peripheral resistance over a long period of time. Following my observations on the clinical case, as reported in the previously mentioned article, I undertook a number of laboratory experiments to substantiate

3. Holman, Emile: Physiology of an Arteriovenous Fistula, Arch. Surg. **7**:64-82 (July) 1923.

the conclusions enunciated therein, that the various cardiac and circulatory phenomena encountered in arteriovenous fistula can be explained on physiologic grounds, with the four variable factors mentioned above as the basis for these phenomena.

In a recent article by Lewis and Drury,[4] exception was taken to this explanation, which was at that time not substantiated by experimental data. The following evidence is now presented in support of the original hypothesis.

Fistulas were produced between the common femoral and common iliac vessels of the right leg, as also between the aorta and vena cava just above the trifurcation. The operatons were performed on a series of dogs under ether anesthesia, morphin ¼ grain (0.016 gm.) having been administered one-half hour before operation. The vessels were exposed over a length of from 4 to 5 cm., and the diameters of the artery and vein measured as they lay in their bed. They were carefully isolated, the tributaries ligated and divided, and the adventitia teased away at the site of the proposed anastomosis. The handling of the vessels incident to this removal of adventitia results in a somewhat diminished caliber, more pronounced in the case of the artery. Small bulldog clamps were applied at the four points and the artery and vein incised along contiguous surfaces for variable lengths, depending on the size of the fistula desired. Junction of the vessels was obtained by suturing the adjacent edges of the longitudinal incisions with a continuous, doubled, Chinese silk suture, threaded in a very fine, straight needle about 1.5 cm. long, the thread being constantly moistened with sterile, liquid petrolatum. In applying this circular suture, it seemed to make little difference in the ultimate healing that followed, if the posterior edges were approximated adventitia to adventitia, and the anterior edges intima to intima. At the conclusion of the anastomosis, the four clamps were removed in the following order: proximal vein, distal vein, distal artery, proximal artery. There was usually brisk bleeding along the line of anastomosis for a moment, but this rapidly subsided.

The opening of such an artificial arteriovenous fistula was always accompanied by the appearance of a palpable thrill, which has become recognized as characteristic of the condition. This thrill was continuous throughout the cardiac cycle, being much accentuated during systole. It could be followed on palpation along the vessels distal to the fistula and also along the proximal vein for variable distances, depending on its intensity. It may be lost within the first two centimeters, or extend as far as 10 cm. from the fistula. Associated with this thrill was a

4. Lewis, T., and Drury, A. N.: Observations Relating to Arteriovenous Aneurysm, Heart **10**:301-389 (Oct. 15) 1923.

loud continuous bruit, which in certain instances of small fistulas could be heard only with the stethoscope, as in animals L2 and L4. A large fistula, as established in Dogs L1, L3 and L28, produced a bruit which, at the time of the operation, could be heard with the unaided ear from 30 to 46 cm. away from the vessels. Sometimes, only the systolic portion of the bruit could be heard with the unaided ear, and then it was a sound resembling the rapid breathing of a small animal. In the larger fistulas, the noise on auscultation was terrific.

A most startling feature of the thrill and the bruit was their complete disappearance on simple closure of the proximal vein, although the blood continued to pour through the fistula into the distal vein. This was a constant observation, and was true if the vein was closed 1 cm. or 10 cm. proximal to the fistula. An explanation was sought in the vibration of the upper or proximal septum between artery and vein, which was set in motion by the swirling, eddying stream of blood as it rushed from the arterial system with its high pressure to the venous system with a much lower pressure. The eddies of arterial blood, which could be seen mixing with the venous blood in the vein proximal to the fistula, were most striking. It was noted, for example, that if, in the process of anastomosing the two vessels, the vein and artery were inadvertently narrowed at the proximal end of the fistula, the thrill was either entirely absent or very feeble.

Observations of the thrill over a period of months also revealed an interesting feature: In the presence of small fistulas, such as in Dogs L2 and L4, there was a definite and well marked thrill at the time of operation. After the elapse of from four to five months, it was observed that the thrill was gradually diminishing in intensity; in the case of Dog L2 it disappeared entirely. This was found on necropsy to have been due to a gradual and finally complete closure of the communication. In Dog L4 it was found to be due to a gradual but not complete closure of the fistula. This spontaneous cure of a small fistula was noted by Reid [5] in his experiments of 1918, and is undoubtedly dependent on the small volume of blood short-circuited through the fistula. In other instances, however, in which large fistulas were made, as in Dogs L1, L3 and L10, the thrill gradually increased in intensity, and at the time of necropsy it was found that there had likewise occurred a gradual dilatation of the vessels proximal to the fistula (Part III), with a correspondingly increased amount of blood flowing through the fistula.

These observations indicate, first, that the presence of the thrill depends on a free return flow of blood to the heart, and, second, that

5. Reid, M. R.: The Effect of Arteriovenous Fistula Upon the Heart and Blood Vessels, Bull. Johns Hopkins Hosp. **31**:43-50 (Feb.) 1920.

the intensity of the thrill is intimately dependent on the volume of this return flow. In our clinical cases, therefore, it is possible to form some estimate of the size of the fistula by the intensity of the thrill and the magnitude of the bruit.

The gradual dilatation of the vessels proximal to the fistula (Part III) must unquestionably increase the volume flow of blood per unit of time through the fistula and back to the heart. In two clinical cases, the dilatation of these proximal vessels was most striking (Figs. 1 and 3). Obviously, under these circumstances the volume of blood flowing through the dilated artery and into the dilated vein must be very great as compared to the flow of blood through the rest

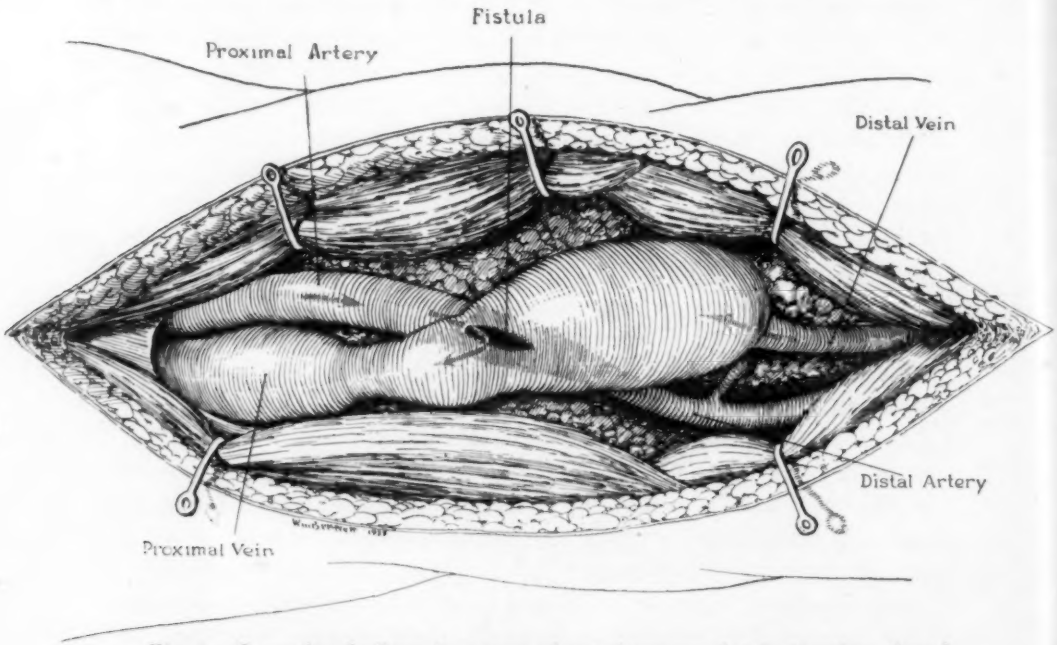


Fig. 1.—Operative findings in a case of arteriovenous fistula showing dilated proximal artery and vein, through which a large volume of blood is being short-circuited to the heart without passing through an intermediate capillary bed.

of the body. In Case 1 (Fig. 1), the proximal artery and vein each had a diameter of 1.8 cm. Distal to the fistula, the artery and vein each measured 0.8 cm. in diameter. The cross section of the column of blood flowing in the artery 1.8 cm. in diameter is determined by the calculation 0.92 by 3.1416, or 2.5 sq. cm., whereas the cross section of the column of blood passing through the artery beyond the fistula was only 0.5 sq. cm. It is apparent that a column of blood 2 sq. cm. in cross section was constantly coursing through the fistula and back to the heart, a volume four times as great as that passing into the leg beyond the fistula.

Starling [6] records the linear velocity in the carotid vessel of the horse as 520 mm. per second during systole and 150 mm. per second during diastole, a mean velocity of 335 mm. per second. The linear velocity in the carotid of a 14 kg. dog is recorded as 241 mm. per second. Assuming that the velocity in the enlarged femoral vessel in this instance was at least equal to that of the dog's carotid, though probably considerably greater, it is safe to say that at least 48 c.c. of blood per second traversed the fistula back to the heart. Starling places the output of the normal heart in man as 60 c.c. per second. Assuming that the output of the greatly dilated heart in this instance was twice that of the normal

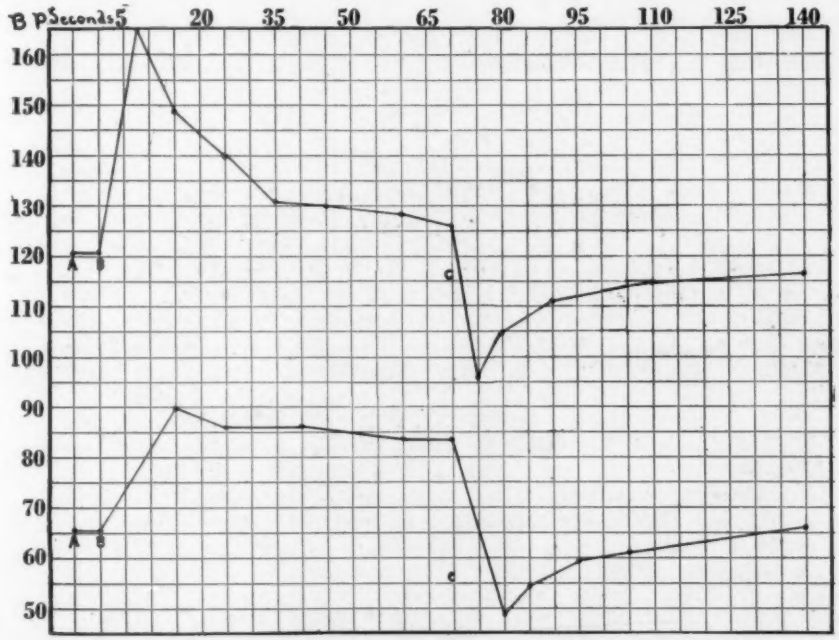


Fig. 2.—Changes in blood pressure (left arm) on closing and opening fistula (in left popliteal space). The lower line represents the fluctuations in diastolic pressure; the upper, in systolic pressure. *A*, fistula open; *B*, fistula closed by digital compression; *C*, fistula opened by release of compression.

heart, it is evident that at least two-fifths of each cardiac output was required to satisfy the demands made by the fistula on the circulating blood.

An exactly similar situation prevailed in the second clinical case (Fig. 3). A column of blood 3.4 sq. cm. in cross section swept down to the fistula. Approximately 1 sq. cm. of this column of blood may be considered as passing into the leg beyond the fistula, thus leaving a

6. Starling: Principles of Human Physiology, J. & A. Churchill Co., 1912.

column of blood 2.4 sq. cm. in cross section to traverse the fistula through the opening 1.8 cm. in diameter. We may assume that at least 1.4 sq. cm. of this column of blood was diverted directly back to the heart through the dilated proximal vein. If the velocity is again estimated at about 240 mm. per second, it is evident that at least 33 c.c. of blood per second was traversing the fistula back to the heart. In this case there was also presented a large, dilated heart, and its output may be estimated at twice normal or about 120 c.c. per second. At least one-fourth its output may therefore be considered as having been diverted through the fistula directly back to the heart, without encountering an intervening capillary bed.

In spite of the necessarily very rough estimates of the amount of blood that traverses the fistula back to the heart, it is evident that the

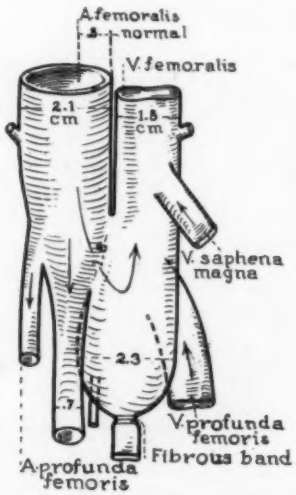


Fig. 3.—Arteriovenous fistula of common femoral vessels, showing marked dilatation of proximal artery and vein, and comparatively small size of vessels distal to fistula. Fistula excised.

introduction of such an abnormal communication into the vascular network introduces thereby a large factor of inefficiency into the circulatory system. It is obvious that the blood flowing through the fistula and back to the heart is a dead loss to the body so far as supplying its nutritional needs are concerned. Provision must be made for the leak, and the demands of the fistula and of the rest of the body must be equally satisfied, or circulatory failure will occur because of poorly nourished tissues.

The abnormal hydraulics of the circulatory system in the presence of a fistula are best understood by consideration from the following point of view: The production of an arteriovenous fistula establishes

two systems of circulating blood—A, heart-artery-capillary bed-vein system, and B, the heart-artery-fistula vein system. Each system may be considered to have a common source of circulating fluid, the heart, but a decided difference in peripheral resistance, depending on the size of the fistula. Blood, like water, will seek the path of least resistance, and in the presence of a fistula large enough to permit the short-circuiting of a considerable portion of the circulating blood, certain adjustments are inevitable if the blood volume flowing through system A is to be sufficient for the survival of the animal. This is only another expression of the necessity of maintaining an adequate general blood pressure, so that a flow of blood will continue through A as well as B. As already indicated, one of the variable factors in the maintenance of blood pressure is the total volume of circulating blood, and an increase of this volume is, according to our theory, one of the inevitable accompaniments of a fistula which permits a large leakage of blood from the arterial into the venous systems.

Our experimental evidence on this point was most surprising and illuminating. The blood volume studies were made according to the method employed by Hooper, Smith, Belt and Whipple.[7] Dr. H. P. Smith performed the first studies in my series and taught me the method. It was my contention that the changes incident to an arteriovenous fistula depended primarily on the size of the fistula and therefore on the amount of blood short-circuited to the heart. Hence, the larger the fistula, the greater would be the compensatory change in blood volume. The following studies were made:

EXPERIMENT 1.—In Dog L2, weighing 13.2 kg., a small femoral fistula 6 mm. long was made, between the right femoral artery and vein, 4 and 5 mm. in diameter. As we shall see (Part II), no changes were ever noted in either the pulse rate or the blood pressure on closure of the fistula, and the necropsy, made June 13, 1923, revealed no change in the size of the heart (Part III), nor any dilatation of the proximal vessels. The preoperative blood volume on October 29 was found to be 1,420 c.c. On May 4, five months after the formation of the fistula, a blood volume determination of 1,410 c.c. was recorded (Fig. 4).

EXPERIMENT 2.—In Dog L4, weighing 14.6 kg., a small fistula 6 mm. long was made, between the right femoral vessels, 3 mm. and 4 mm. in diameter. Only very slight changes in blood pressure and pulse were noted on opening and closing the fistula, and these were of a very transitory nature. At necropsy, four months after the production of the fistula, the heart showed a slight dilatation of the right half, there was slight visible dilatation of the proximal vein, and a suggestive dilatation of the proximal artery not visible or detectable by actual measurement but brought out by injecting the vessels with sodium iodid and by taking a roentgen-ray photograph (Part III). A preoperative blood volume estimation of 1,470 c.c. was made Dec. 19, 1922, and June 14, 1923, a volume of 1,630 c.c. was determined, representing a small increase (Fig. 4).

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7. Hooper, C. W.; Smith, H. P.; Belt, A. E., and Whipple, G. H.: Blood Volume Studies, Am. J. of Physiol. **51**:205-220 (March 1) 1920.

EXPERIMENT 3.—In Dog L3, weighing 15 kg., a large fistula 3.4 cm. long was established Nov. 9, 1922, between the right femoral artery and vein, measuring 4 and 6 mm., respectively. As it will be shown later (Parts II and III), this dog showed a rather marked increase in blood pressure and a retardation of pulse rate when the fistula was closed, with a definite increase in the size of the cardiac area, and also a dilatation of the proximal vessels. On November 20, a blood volume estimation of 1,400 c.c. was made, and by June 12 the volume had increased to 1,680 c.c. On June 16, the fistula was excised, and on the following day, a blood volume determination of 2,170 c.c. was made. This was repeated four days later, and a volume of 2,150 c.c. was recorded; twenty-five days later, it had, however, again dropped to 1,520 c.c., and in the following October, 1923, it was estimated at 1,370 c.c. November 9, a second fistula was performed

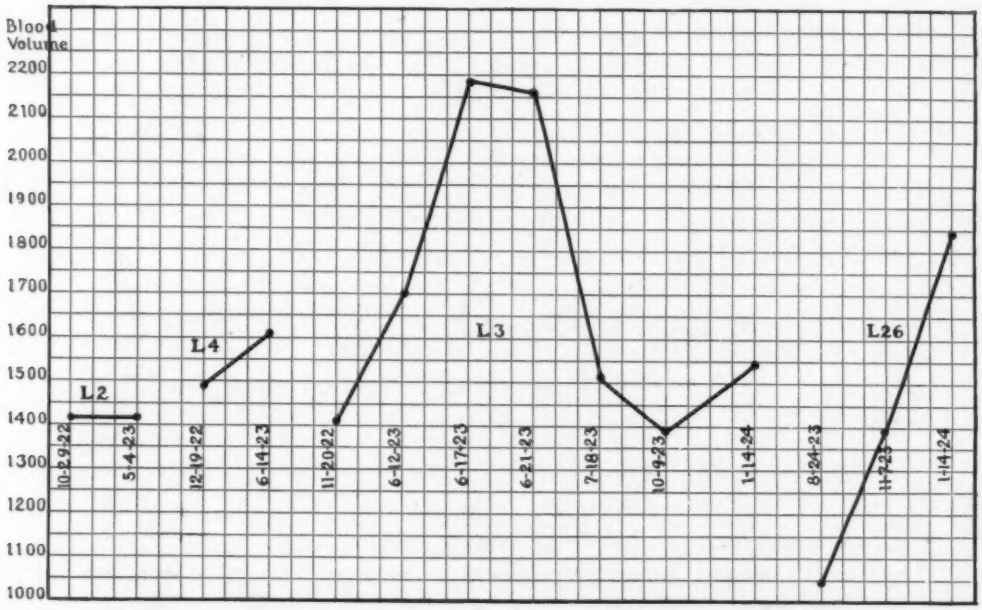


Fig. 4.—Blood volume determinations: (*a*) constant blood volume in L2, with a small fistula that healed spontaneously; (*b*) slight increase in L4, with a small fistula; (*c*) marked increase in L3, with a larger femoral fistula followed by subsidence of this increase after excision of the fistula; (*d*) marked increase in L26, with a large iliac fistula.

between the iliac vessels, and on Jan. 14, 1924, a blood volume determination of 1,530 c.c. was made (Fig. 4).

EXPERIMENT 4.—In Dog L 26, weighing 12 kg., a large fistula 3 cm. long was established on Aug. 29, 1923, between the right common iliac artery and vein measuring 4 and 6 mm., respectively. A preoperative blood volume estimation of 1,039 c.c. was made Aug. 24, 1923. On November 7, a blood volume of 1,340 c.c. was determined. January 14, a volume of 1,830 c.c. was recorded (Fig. 4). January 25, the dog was killed, and the necropsy revealed a definite enlargement of the vessels proximal to the fistula, and a very greatly enlarged

heart (Part III), particularly the right half. Making allowance for an increase in weight during this time from 12 to 15 kg., there is still a marked relative increase in blood volume.

It is to this increase in total blood volume that we ascribe the fact that the low pulse noted on closure of a large, long-standing fistula is maintained until the fistula is again opened. Obliterating the abnormal communication restores the circulatory system to its original status of heart-artery-capillary bed-vein. That portion of the blood volume which formerly leaked through the fistula now flows around the system A, all the vessels of which, including the aorta, become distended by the inflow of a volume of blood which has increased in amount since the formation of the fistula, the amount of increase depending on the size of the fistula. In addition, the mean peripheral resistance is also greatly increased by closure of the artificial communication that restores the normal capillary channels. The sudden increase in these two important factors concerned in the maintenance of blood pressure, namely, blood volume and peripheral resistance, should, theoretically, produce an increased blood pressure, and, practically, all our records, both clinical and experimental, have demonstrated this to be so. Clinically, in our first case, there was a transitory sudden rise from 120 to 160 when the fistula was closed, falling within a few seconds to 126, where it remained until the fistula was opened again (Fig. 2). In our second case, closure of the fistula raised the pressure from 116 to 180 for two beats only, after which it fell rapidly to 140, where it remained until the fistula was opened (Fig. 5). This tremendous, initial rise in blood pressure would almost instantly be rectified theoretically by adjustments in the remaining two variable factors concerned in the maintenance of blood pressure, namely, the cardiac output or rate of contraction, and the capacity of the system as influenced by dilatation of the peripheral vessels. With reference to the adjustment in pulse rate, all our observations demonstrate a remarkable retardation of rate on closure of the fistula, less pronounced in recent fistula, but gradually becoming more evident as the amount of blood flowing through the fistula is increased, and dilatation of the proximal vessel leading to the fistula has occurred. By closure of the fistula, this increased volume of blood previously short-circuited through system B is suddenly thrown into system A, and there is an immediate compensatory adjustment for the increased blood pressure by a retardation in heart rate. Opening the fistula again diverts this increased volume of blood into the capacious heart-artery-fistula vein system, blood pressure drops, and the pulse rate returns to its previous higher level.

With reference to the adjustment obtained by an increase in the capacity of the system as determined by peripheral dilatation, Meeker and

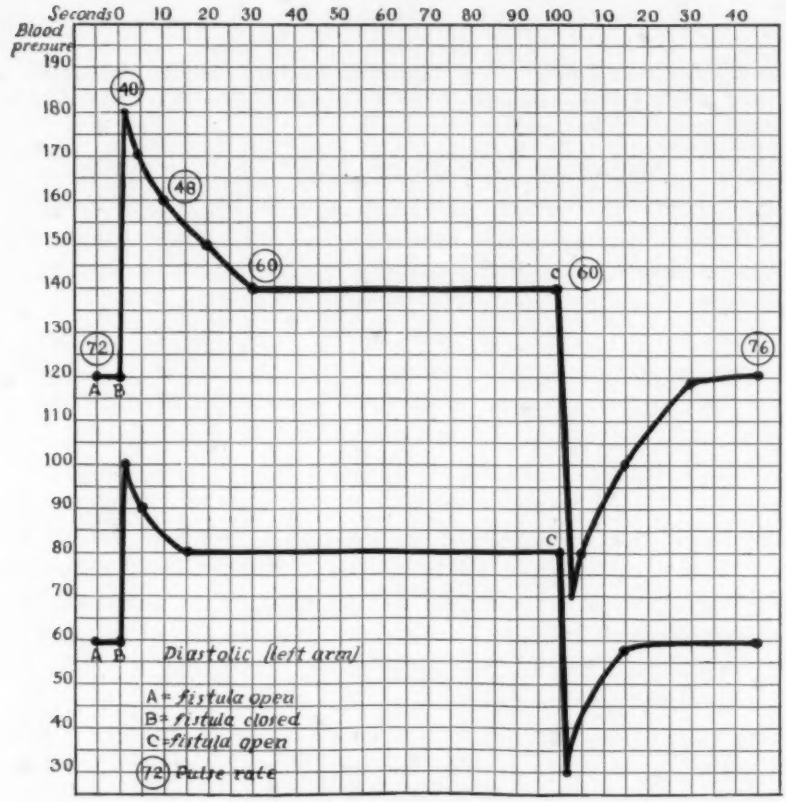


Fig. 5.—Fluctuations in general blood pressure (left arm), on closing and opening fistula between the right common femoral vessels, accompanied by marked pulse variations (same case as Figure 3).

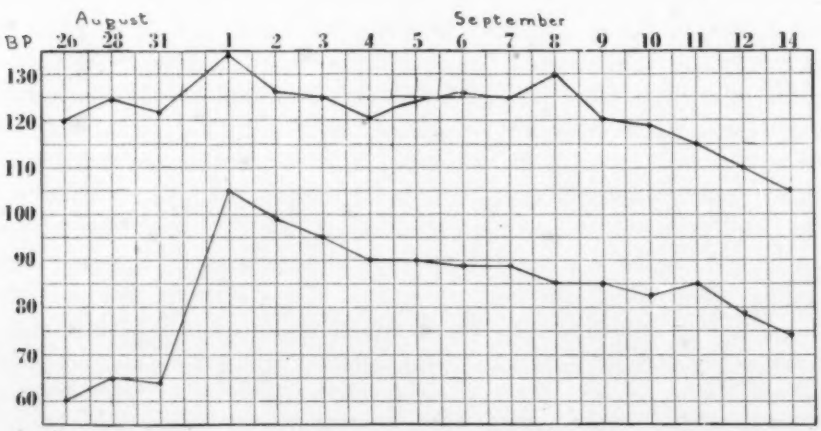


Fig. 6.—Blood pressure readings: (*a*) high pulse pressure in presence of popliteal arteriovenous fistula; (*b*) marked increase in both systolic and diastolic blood pressure levels following repair of fistula Aug. 31, 1922; (*c*) gradual subsidence of this high level with permanent increase in diastolic level, and a permanent decrease in pulse pressure.

Eyster [8] have recently shown that a considerable increase in blood volume may be added to the circulation without evident increased content of the arteries, without noticeable stasis in the venous cisterns, without cardiac dilatation, but with very definite photomicrographic evidence of a distention of the peripheral vessels, the capillaries and venules.

In spite of these adjustments in pulse rate and a probable dilatation of the peripheral bed to accommodate the increased blood volume, we note in our clinical cases a well sustained increase in general blood pressure considerably above the normal level, maintained as long as the fistula remained closed. In our first clinical case, a blood pressure of 120/66 was increased to 126/88 throughout closure of the fistula (Fig. 2), in spite of a pulse rate of from 35 to 40. In our second case,

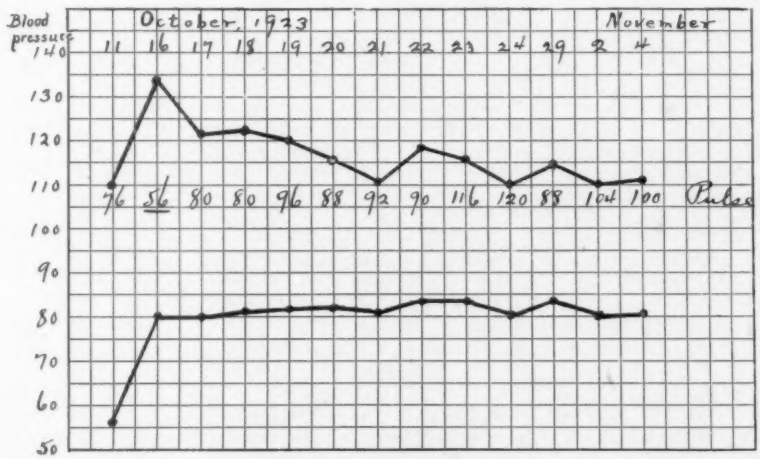


Fig. 7.—Blood pressure readings: (*a*) high pulse pressure in presence of arteriovenous fistula of common femoral vessels; (*b*) marked increase in systolic and diastolic levels following excision of the fistula Oct. 15, 1923; (*c*) gradual subsidence of the high systolic level, with a permanent increase in diastolic level, and a permanent decrease in pulse pressure; (*d*) marked retardation of pulse rate on the day following excision of fistula, accompanying the temporarily increased blood pressure.

the pressure of 120/56 remained increased to 140/80 until the fistula was opened, in spite of a pulse rate of 60 (Fig. 5). What happens when the fistula is permanently closed by operation?

In the first case, the day following the repair of the fistula, the blood pressure readings were 135/105 (Fig. 6), as compared to a preoperative

8. Meeker, W. J., and Eyster, J. A. E.: The Effect of Plethora and Variations in Venous Pressure on Size and Output of the Heart, Am. J. Physiol. **61:** 186-202 (June) 1922.

reading of 120/66. Two days later, the pressure had dropped to 124/96, nine days later to 120/85, and two weeks later to 110/78, approximately a normal figure, with the diastolic considerably increased over the preoperative level. The second clinical case exhibited a similar picture (Fig. 7): a preoperative blood pressure of 110/56 was increased the day following excision of the fistula to 130/80. There was then a gradual subsidence of systolic pressure to the preoperative level of 110 to 120, while the diastolic level remained around 76 to 80.

An exact duplication of this clinical picture was obtained experimentally in Dog L3 (Fig. 8), in which a preoperative blood pressure (unanesthetized animal) of 200/90 was obtained. Seven months after

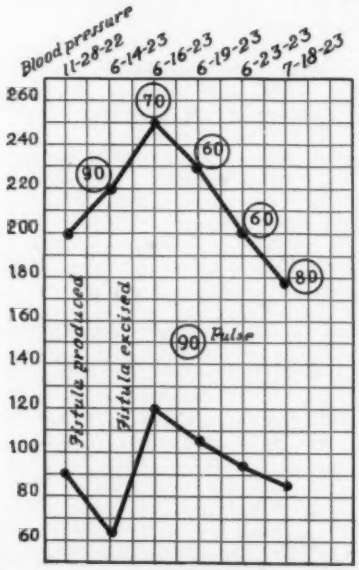


Fig. 8.—Experimental duplication of the clinical observations noted in Figures 6 and 7, by establishing a fistula between the common femoral vessels, Nov. 28, 1922, and excising it, June 15, 1923.

the formation of a large femoral fistula, a pressure of 220/65 was recorded. The fistula was excised, and on the following day the pressure was 250/120; four days later it was 230/105; six days later, 200/95, and a month later 176/86. A remarkably slow pulse of from 60 to 70 was maintained for several days following the operation during the period of very high blood pressure, but this soon returned to a normal rate (Part II).

The gradual readjustment in blood pressure and pulse rate following the permanent closure of the fistula, as noted both clinically and experimentally, can be attributed to only one of our four variable factors concerned in the maintenance of blood pressure, namely, total blood volume,

and our experimental evidence very strongly sustains this explanation. It is to this increase in total blood volume that we must ascribe Branham's phenomenon, which, as we shall show (Part II), gradually develops following the formation of a fistula, and which gradually subsides again following the excision of the fistula. The strange and inexplicable observation in the Matas clinic case is dependent on the same factor—the low blood pressure on the normal side was taken with the fistula open, and the high pressure on the affected side was necessarily obtained with the fistula thrown out of the circulation through constriction of the femoral vessel by the blood pressure apparatus. With the fistula closed, the general blood pressure was raised, because the blood previously short-circuited through the fistula was diverted into the normal circulatory channels.

Some recent critical reviews [9] of our present methods of blood volume determination indicate the necessity of interpreting one's results as rather inaccurate so far as actual absolute volume is concerned. That comparable observations on the same individual may be made with our present methods, these authors are willing to admit. The results we have obtained are striking and suggestive, and may be interpreted with some degree of assurance that changes do occur in blood volume in the presence of a fistula, and this in the direction of a definite increase, which may be rectified by excision of the fistula.[10]

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9. Lamson, P. D., and Rosenthal, S. M.: The Inadequacy of Our Present Blood Volume Methods, Am. Jour. of Physiol. **63**:358-367 (Jan.) 1923.

10. The variations in blood volume demonstrated here open up a wide field of speculation as to the nature of blood volume control in the body, and its importance in numerous other clinical entities which remain unexplained. By what mechanism is blood volume maintained fairly constant under such varying blood conditions as exist in primary anemia and polycythemia vera? What mechanism in the body determines the excretion of fluid under varying conditions of desiccation and hydremic plethora? Are blood volumes identical per body weight in persons with blood pressures of 120/70 and 240/120? What is responsible for so-called essential hypertension? May it not be due solely to an increased blood volume, the result of an impairment of the mechanism for its control? May not this impairment be the first step in the development of so-called chronic cardiovascular-renal disease? I venture the hypothesis that a heart never dilates or hypertrophies because of the supposed increase in effort necessary to push the blood through a distant, increased, peripheral resistance as in the case of a diseased kidney, but that it dilates and hypertrophies because of an increased total blood volume and an increase in the effort necessary to propel forward this increased blood flow through its chambers, the increase in blood volume being the result of a diseased function of the organs concerned in its control, more particularly, perhaps, the kidney.

The perfection of an accurate means of determining blood volume, so that comparable observations between individuals may be made, should bring about a solution of these interesting problems.

SUMMARY

The magnitude of the bruit, and the intensity of the thrill in arteriovenous fistula depend on the size of the fistula and the free return flow of blood to the heart. Both phenomena disappear on simple closure of the vein proximal to the fistula even though blood continues to pour through it toward the periphery. The thrill is ascribed to the vibration of the septum between artery and vein produced by the eddying blood as it sweeps under high pressure from the arterial into the venous systems.

The production of a fistula introduces a large factor of inefficiency into the circulatory system, particularly with reference to the maintenance of blood pressure. The two systems, A, heart-artery-capillary bed-vein, and B, heart-artery-fistula vein, must be adequately supplied with a circulating medium, or the blood pressure will fall below a level compatible with life. The compensatory adjustment that follows the introduction of a fistula is according to the experimental evidence here presented, an increase in total blood volume, the increase depending on the size of the fistula and its duration.

The effect of this increase in total blood volume on pulse and blood pressure as well as on the size of the heart and blood vessels will be considered in subsequent papers.

# EXPERIMENTAL STUDIES IN ARTERIOVENOUS FISTULAS

## II. PULSE AND BLOOD PRESSURE VARIATIONS *

EMILE HOLMAN, M.D.
Resident Surgeon, Peter Bent Brigham Hospital
BOSTON

ASSISTED BY
ALFRED C. KOLLS, M.D.
Associate in Physiology, Johns Hopkins Medical School
BALTIMORE

In Part I, the abnormal hydraulics of the circulatory system in the presence of an arteriovenous fistula was discussed and the following points emphasized: (1) An arteriovenous fistula introduces into the normal circulation consisting of heart-artery-capillary bed-vein, a second system of circulating blood consisting of heart-artery-fistula vein, the latter with a markedly diminished peripheral resistance; (2) the existence of four variable factors to maintain the normal circulation of blood at a definite blood pressure level, namely: (a) cardiac output or pulse rate, (b) total capacity of system as determined by contraction or dilatation of the vessel walls, (c) total volume of circulating blood, and (d) peripheral resistance. To compensate for the lowered peripheral resistance in the presence of a fistula, and to provide for the leakage of blood through the area of lowered resistance, thereby maintaining an adequate blood pressure level, an increase in total volume of circulating blood was postulated, and experimental evidence of such an increase was presented.

Evidence was also presented to show that this increase in total blood volume depended on the size of the fistula. It is the purpose of this article to demonstrate the very intimate relationship that exists also between the size of the fistula and the pulse and blood pressure variations which occur on opening and closing the abnormal communication. Our experimental evidence was obtained by means of an Erlanger apparatus adapted for use on a dog,[1] the pressures being obtained by means

* From the Laboratory of Surgical Research, Harvard Medical School, and the Hunterian Laboratory of Experimental Surgery, Johns Hopkins Medical School.

1. Kolls, A. C.: An Indirect Method for the Determination of Blood Pressure in the Unanesthetized Dog, J. Pharmacol. & Exper. Therap. **15**:443-452 (July) 1920.

of a specially prepared cuff applied to the shaved leg of the animal. As examples of the data obtained from our experiments, the following observations are here recorded:

EXPERIMENT 1.—In Dog L1, operated on Oct. 16, 1922, the right femoral vessels were exposed, the artery measuring 3 mm. in diameter and the vein 4 mm. An elongated, oval communication 3.5 cm. long and approximately 2 mm. wide was made between the two vessels. Before removal of the clamps, the blood pressure in the left femoral artery was 204/124, the pulse 148, and the respirations from 40 to 44. The opening of the fistula resulted in a drop of general pressure, recorded in the left femoral artery, to 176/80, with an acceleration of pulse to 214 and 224. The respirations had also increased slightly to 48 and 52. The stream of arterial blood was seen to pass through the fistula and into the proximal vein, although a small but definite pulse could be felt in the artery below the opening. When the fistula was compressed and the communication thus obliterated, the blood pressure recovered to 208/110 with a corresponding fall in the pulse rate to 180. When the fistula was opened, the pressure again fell to 200/80. The pressure in the right femoral artery below the fistula was recorded as 135/30. The wound was closed and healed by first intention.

Seven days later, compression of the fistula resulted in definite though very slight transitory changes in the general pressure (Fig. 1 *A*), with a variation in the pulse rate from 102 with the fistula closed, to 120 with the fistula open. On November 2, these changes manifested on the opening and closing of the fistula were slightly more pronounced (Fig 1 *B*), and by November 23, they had become even more striking (Fig. 1 *C*). The dog died November 29 of distemper and pneumonia, and the necropsy revealed a marked increase in the caliber of the vessels proximal to the fistula (Fig. 2).

The progressive character of the variations in pulse and blood pressure occurring on the closing and opening of the fistula is undoubtedly dependent on this gradual dilatation of the vessels, a dilatation which in turn is entirely dependent on a gradual increase in the volume of blood passing through the heart-artery-fistula vein system, this increase also producing *pari passu* an increase in the total volume of circulating blood (Part I).

As Branham observed in his clinical case, the retardation of the pulse rate "gradually" became more marked. Our experiments show that for some days following the formation of a fistula, closure produced a transient fall in pulse rate and a transient rise in pressure. The longer the fistula remained, however, the more pronounced and more prolonged became these changes, until finally they persisted as long as the fistula remained closed, as in our clinical cases. This phenomenon seems definitely linked with the gradually increasing total volume of circulating blood.

Interesting observations illustrating the phenomena that occur in the presence of a large fistula were obtained:

EXPERIMENT 2.—In Dog L3 a communication 3.4 cm. long was made November 29 between the right femoral artery and vein, measuring 4 mm. to 6 mm.,

Fig. 1 (Experiment 1, Dog L1).—Kymographic records showing the progressive character of the changes that occur on the closing (c) and opening (o) of large fistula: *A*, seven days following formation of fistula; *B*, seventeen days following formation of fistula; *C*, thirty-eight days following formation of fistula.

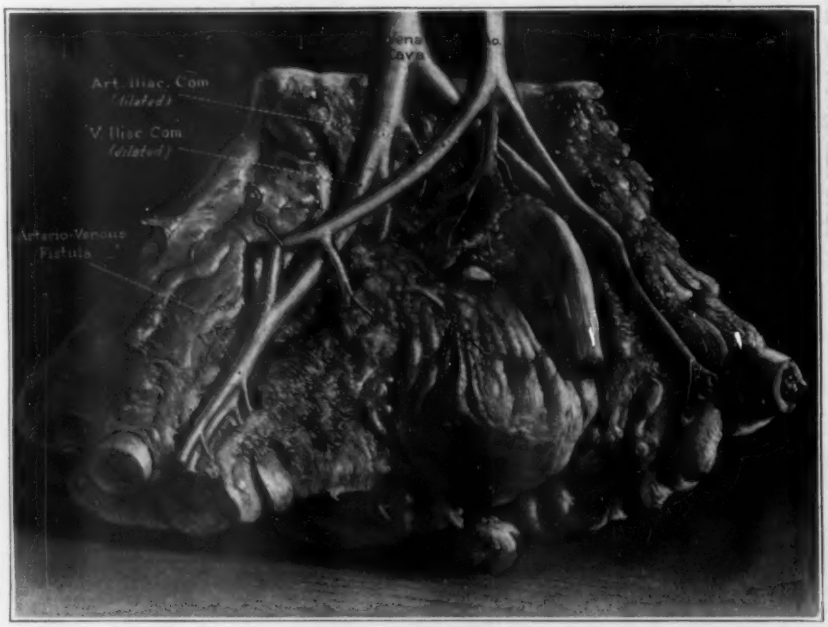


Fig. 2 (Experiment 1, Dog L1).—Dilatation of proximal vessels, forty-four days after the formation of fistula. Drawing shows the region in which these fistulas were produced, and the character of the linear anastomosis employed.

respectively. The general blood pressure in the left femoral artery was 200 systolic and 90 diastolic before operation. When the fistula was opened it dropped to 190/70, with a recovery to 205/80 on closure. Figure 3 *A* illustrates the profound changes in the general blood pressure occurring on the operating table as soon as the conditions at the fistula were altered, a sudden, sharp increase when the fistula was closed, and a sudden fall when the fistula was opened. This record was taken at the end of a long operation attended by considerable shock. The pulse rate was exceedingly rapid, 224 per minute. On December 7, nine days after the anastomosis, definite changes were recorded on the opening and closing of the fistula, the pulse dropping from 78 to 66, and the blood pressure increasing at the same time from 170/60 to 185/75 (Fig. 3*B*). April 4, a marked increase in pressure, with a fall in pulse from 72 to 42 was recorded when the fistula was closed (Fig. 3*C*). As the chart indicates (Fig. 4), these changes gradually become more pronounced, and June 14 a drop

Fig. 3 (Experiment 2, Dog L3).—Effects of opening and closing a fistula: *A*, immediately after its formation; *B* and *C*, progressive character of the changes; the retarded pulse rate in *C* was maintained as long as the fistula remained closed.

in pulse rate from 90 to 48 was recorded, together with an increase in blood pressure from 205/60 to 240/95 when the fistula was closed.

June 15, an operation for the cure of the fistula was performed; the vessels in the groin were exposed and isolated above, below, and including the fistula. The thrill in the exposed vessels was intense, and the swirling arterial blood could be seen flowing back to the heart through the vein above the fistula. Closure of the proximal artery, of course, abolished the thrill, but closure of the proximal vein abolished it equally well. The conditions found at the site of the fistula were in accord with previous observations. There was a definite dilatation of both artery and vein proximal to the communication (Part III). The vessels were ligated above and below the fistula and the communication excised.

The pulse before operation was 166; after ligation of the vessels, 144, and at the end of operation, 140. These variations were the reverse of those noted at the first operation in November, ten months previously, when the artificial communication was established, the pulse at that time having increased during the operation from 140 to 228. Following the excision of the fistula, the blood pressure rose from 206/60 to 250/110 and remained there for twenty-four hours. During the first few days following excision, the pulse rate remained unusually

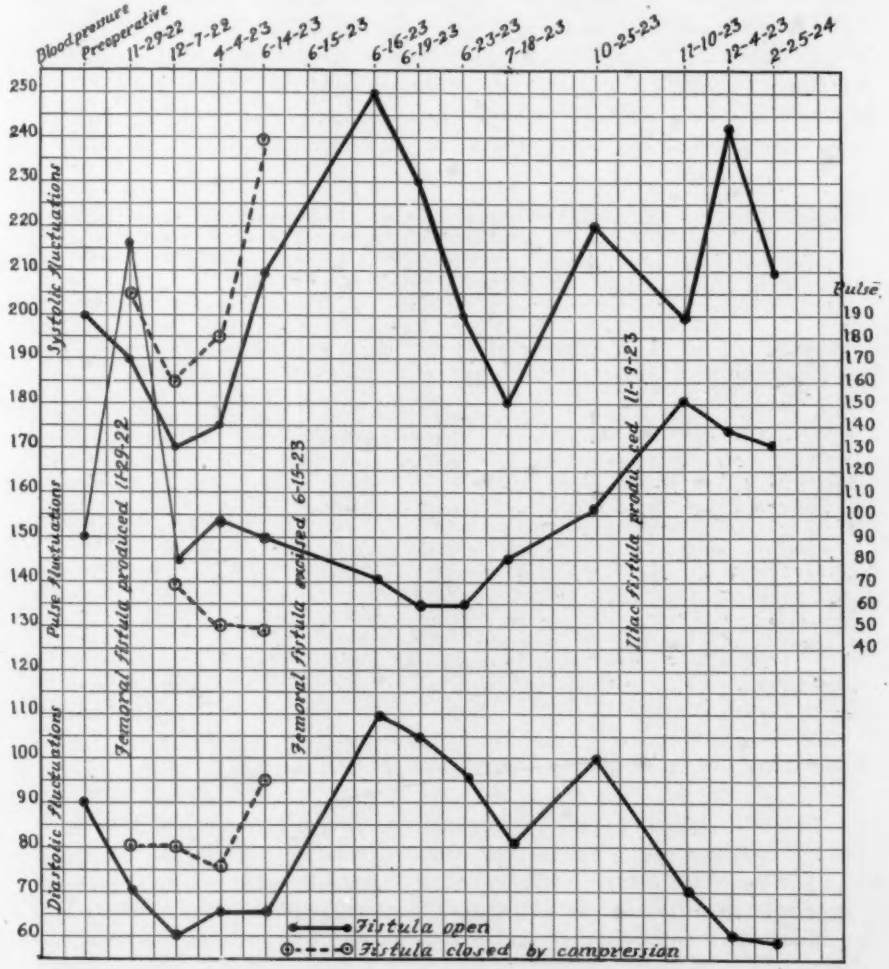


Fig. 4 (Experiment 2, Dog L3).—The blood pressure and pulse variations accompanying a fistula of long duration. The following observations may be made: (1) increased pulse pressure in presence of fistula, reduced by excision of fistula, and again increased by formation of iliac fistula; (2) increase in systolic and diastolic levels, with retardation of pulse by compression of femoral fistula; (3) marked increase in pressure and fall in pulse following excision of fistula, with a gradual subsidence in pressure and increase in pulse; (4) rapid pulse and low diastolic pressure in presence of iliac fistula.

low, increasing gradually from 60 to 80, accompanied by a gradual subsidence of the high blood pressure. Three days after operation, a pressure of 230/105 was recorded, with a pulse of 60; eight days later it was 195/100, with a pulse of 60; a month later, the pressure had again dropped to a normal level of 176/82, with a pulse of 80. The full significance of these variations was discussed in a previous paper and was attributed to a gradual decrease in volume of circulating blood (Part I). On Oct. 25, 1923, a blood pressure of 220/98 was obtained with a pulse of 102. November 9, the right common iliac vessels were exposed, and an anastomosis 2.8 cm. long was made. The pulse rate before operation was 84; before opening the fistula it was 180, and immediately after opening the fistula, 210. A marked bruit could be heard 38 cm. from the anastomosis, entirely controlled by simple closure of the proximal vein. The pulse at the end of the operation was 220. A resounding "pistol shot" could be

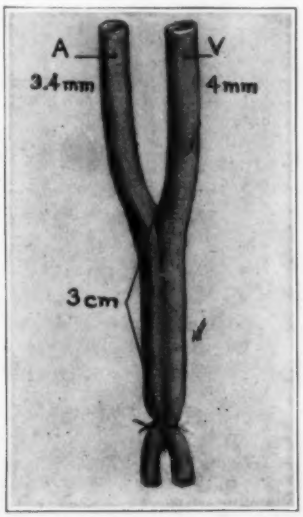


Fig. 5 (Experiment 3, Dog L10).—Large femoral fistula established Feb. 28, 1923; artery and vein distal to fistula ligated.

heard on auscultation over the left femoral artery. On November 10, a pulse of 150 was recorded with a pressure of 196/70; December 4, a pressure of 244/60 with a pulse of 130 was recorded, indicating a markedly increased pulse pressure comparable to that noted in aortic insufficiency. The similarity of these two conditions has been emphasized by Lewis and Drury.[2] A roentgen-ray examination of the heart made on this day (Part III) also showed a definite cardiac enlargement. January 12, the following note was made: The pulse was 138, and the heart was beating vigorously and tumultuously against the chest wall. There was no swelling of the leg or of the abdominal wall, and no prominence of the veins of the leg or of the abdominal wall. A loud pistol shot was heard over the left femoral artery, and a loud bruit was heard over the whole abdomen

2. Lewis, T., and Drury, A. N.: Observations Relating to Arteriovenous Aneurysm, Heart **10**:301-389 (Oct.) 1923.

extending to the xiphisternum. February 25, it was observed that the dog was failing. A pulse of 132 with a pressure of 210/58 was recorded, the diastolic even lower than that previously noted. The dog was killed March 10, 1924, when it was quite ill with bronchial sepsis.

These observations demonstrate very clearly the lowered diastolic pressure in the presence of a fistula, but they also illustrate the fact that the systolic pressure may be increased, the essential difference being a greatly increased pulse pressure following the production of an arteriovenous fistula. The repair of a fistula, as observations on Dog L3 (Experiment 2) indicate, causes a well marked reduction in pulse pressure, the diastolic pressure remaining at a permanently higher level than that noted in the presence of a fistula, and at approximately the same level noted before the production of the fistula.

A third illustration of the effects of a large fistula was obtained in Dog L10:

EXPERIMENT 3.—In Dog L10, the femoral artery and vein, measuring 3.4 and 4 mm. in diameter, were united by a linear oval anastomosis 3.1 cm. in length (Fig. 5). Both vessels were ligated at the distal extremity of the fistula, thus producing in effect an end-to-end anastomosis, the object being to secure if possible a more rapid dilatation of the proximal vessels and heart. A marked thrill and an audible bruit were present at the end of the operation. Thirty-five days later, the changes in pulse rate and blood pressure brought about by closing the fistula were quite conspicuous (Fig. 6), and at necropsy two months later, the vessels were considerably dilated proximal to the fistula (Fig. 7).

Dog L28 also furnished some instructive data (Fig. 8):

EXPERIMENT 4.—Sept. 18, 1923, a preoperative blood pressure of 216/106, with a pulse of 84 was obtained in Dog L28. October 22, an anastomosis between the right common iliac vessels was established, the artery and vein measuring 5 and 7 mm., respectively. The fistula was 2.5 cm. long. A pulse rate of 120 increased to 140 during the operation. When the fistula was opened, the pulse rate immediately rose to 210 where it remained during the rest of the day. On the following day, within less than eighteen hours, the right leg was greatly swollen; but the dog was able to jump out of her cage, limping and dragging the right leg. The blood pressure was very low, 110/60, and the pulse rate still remained around 200. The subsequent blood pressure and pulse readings are recorded in the chart (Fig. 8). The pressures in the right leg below the anastomosis were always very low, approximately 50/20, indicating a fairly complete diversion of blood through the fistula.

The importance of these observations lay in the marked reduction in both systolic and diastolic levels immediately following the production of a fistula, and in the gradual recovery of the systolic pressure to normal, with the persistence of a low diastolic pressure throughout our observations. There is, therefore, a very evident and marked increase in pulse pressure in the presence of an experimental arteriovenous fistula, which has already been noted in our clinical cases; this increased pulse

Fig. 6 (Experiment 3, Dog L10).—Effect on blood pressure and pulse obtained by the opening and closing of a large femoral fistula, thirty-five days after its formation.

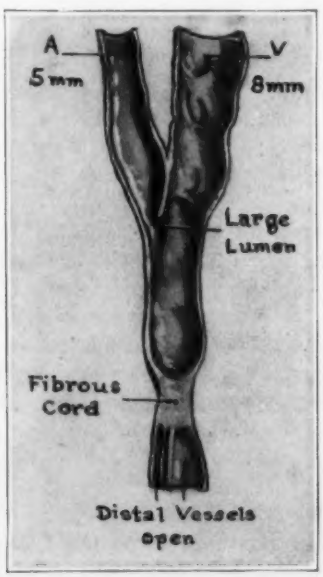


Fig. 7 (Experiment 3, Dog L10).—Definite dilatation of proximal artery and vein disclosed at necropsy, June 11, 1923.

pressure is apparently due not only to a fall in diastolic pressure, but also to a slight increase in systolic pressure (Experiment 2, Dog L3). The latter phenomenon is probably associated with the enlargement of the heart, with the increased venous filling and the increased total blood volume that develop in the presence of an arteriovenous fistula. This animal, Dog L28, was killed Jan. 29, 1924, and a marked enlargement of the heart and of the proximal vessels was revealed (Part III).

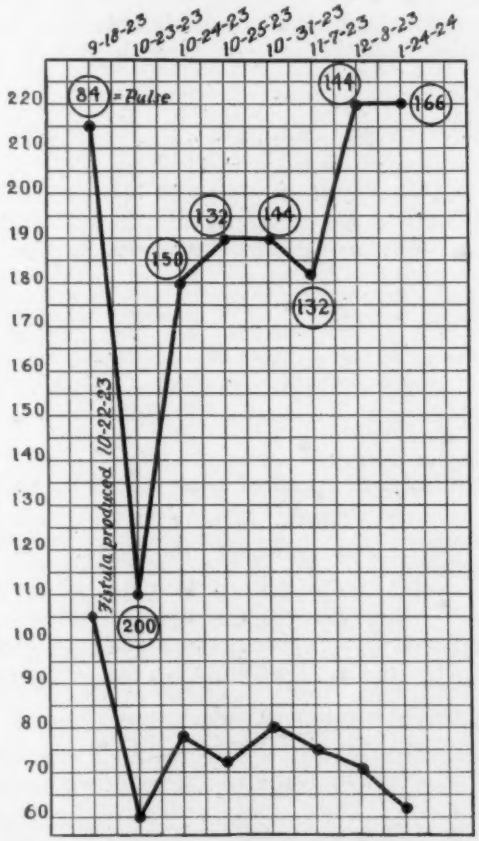


Fig. 8 (Experiment 4, Dog L28).—Blood pressure and pulse readings, illustrating: (1) tremendous fall in blood pressure immediately after the formation of an iliac fistula, with a very rapid pulse; (2) recovery of systolic pressure, but a permanent reduction in diastolic pressure, and a permanent increase in pulse rate; (3) the extent of these variations as considerably more conspicuous in an iliac fistula than in a femoral fistula (Fig. 4).

EXPERIMENT 5.—Additional evidence of the introduction of a fistula into the vascular network was obtained by observations on Dog L27, in which the aorta and vena cava were anastomosed just above the trifurcation. During the first part of the operation, the pulse rose to 180, and when the fistula was opened

the pulse immediately rose to 240. Clamping the proximal artery resulted in an immediate fall in the pulse rate to 200; opening the proximal artery reestablished the fistula, with acceleration of the pulse to 240. The important observation, however, was that closure of the proximal vein also caused the pulse rate to drop to 200, with an acceleration to 240 when the vein was opened. Clinically, this was also observed during an operation for the excision of a large femoral fistula. Simply closing the vein proximal to the open fistula caused the pulse rate to drop from 108 to 84.

The influence of venous filling on the well-functioning, unimpaired heart muscle has been the subject of considerable experimentation by our most eminent physiologists.

Bainbridge [3] writes as follows:

> Krogh has pointed out that when the venous inflow to the heart is so large that the right ventricle contains as much blood as it can hold before systole occurs (he terms this an adequate supply to the heart) the output of the heart per minute depends chiefly on its frequency. . . . The quickening of the heart, which occurs in plethora or hydremic plethora, must be of value not only in increasing its output when the venous filling of the heart is very rapid, but also in lessening the risk of its becoming so engorged that it becomes mechanically inefficient. . . . The heart is therefore provided with a reflex mechanism, whereby, when the venous filling is increased, the circulation can be maintained by the more rapid transference of blood from the venous to the arterial system, and the risk of excessive dilatation and of failure of the heart is lessened.

In our experimental animals with large fistulas, opening the fistula always resulted in a rise in pulse rate, as we have already shown, with a corresponding fall in rate on closing the fistula. As will be demontrated later (Part III), opening the fistula was also accompaned by a dilatation of the heart, indicating that there must of necessity be an "adequate" supply to the heart, in the sense that Krogh intends. Obviously, therefore, the output of the heart is necessarily increased commensurate with the increase in pulse rate.

The observation that the thrill and bruit were obliterated by closure of the proximal vein prompted the following procedure:

> EXPERIMENT 6.—In Dog L11, the femoral vessels were exposed, the diameter of the artery and vein measuring 3.8 mm. and 4 mm., respectively. A fistula 3 cm. long was made between the two vessels. The marked thrill and bruit, observed immediately after the removal of the four clamps, were completely abolished by ligation of the vein proximal to the fistula. The blood from the proximal artery passed almost equally into the distal artery and through the fistula into the distal vein. At no time after the operation was a thrill palpated or a bruit heard at the site of the anastomosis, in spite of an observed pulsation in one of the superficial veins of the leg below the level of the fistula. Within seven days after the operation there was very marked

3. Bainbridge, F. A.: The Influence of Venous Filling upon the Rate of the Heart, J. Physiol. **1**:65-84. 1915-1916.

swelling of the right thigh with complete disability of this leg, a condition which was no doubt the result of filling the venous side with arterial blood. This swelling gradually subsided and within six weeks had entirely disappeared. Pressure over the site of the fistula never produced any effect on the pulse rate or on the general blood pressure. It seems clearly evident that these pressure and pulse variations are entirely dependent on a free return flow to the heart; from our observations, it is apparent that the greater this return flow the more pronounced are the changes. On June 16, 106 days after the operation, the dog was killed. The femoral vessels on both sides were isolated, and a careful examination revealed no alterations in the vessels of the affected side. The vein proximal to the fistula had become obliterated from the fistula to the point of ligature of the vein, and there was no evidence of a communication between the artery and vein (Part III).

From these findings, it is apparent that the unimpeded return flow to the heart is not only necessary for the production of the thrill and bruit so characteristic of the arteriovenous fistula, but that it is also directly responsible for the dilatation of the vessels proximal to the fistula. As a corollary to this fact, no pulse or blood pressure variations were ever noted in response to the opening and closing of the fistula.

Further observations on dogs in which small fistulas had been made yielded results that differed quite markedly from those noted in the presence of large fistulas.

EXPERIMENT 7.—In Dog L2, the femoral artery and vein on the right measured 4 mm. and 5 mm., respectively. An opening between the two vessels about 6 mm. long and 2 mm. wide was made. The characteristic thrill was present as soon as all the clamps were released, but it was only of feeble intensity as compared to that present with larger communications. There were no demonstrable changes in blood pressure or pulse on closing and opening the fistula, either immediately after operation or on any subsequent occasions (Fig. 9). The anastomosis was performed on Jan. 12, 1923. On March 26, the gradually disappearing thrill was noted for the last time, and at necropsy, two months later, the fistula was found to have entirely healed (Part III).

Somewhat similar observations were made on Dog L4.

EXPERIMENT 8.—In Dog L4, a slitlike anastomosis 6 mm. long was made between the right femoral artery and vein, measuring 3 and 4 mm., respectively. At the end of the operation a definite thrill was present, which persisted throughout life. A definite drop in pressure occurred when the fistula was opened, but at no time subsequently were there any marked changes present on the closing and opening of the fistula, although very slight transitory variations were occasionally observed (Fig. 10). The dog was killed six months after the operation, and the communication was still present. There was a slight dilatation of the proximal vein, and a suggestion of dilation in the right side of the heart, but no visible alteration in the size of the artery as compared to the left side (Part III).

These examples reveal a very definite relationship between the size of the fistula, the blood pressure changes, and the visceral changes.

In the course of investigating the immediate results attending the opening of an arteriovenous fistula, it was observed that the resultant

Fig. 9 (Experiment 7, Dog L2).—No alterations in pulse or blood pressure in a small fistula on the closing and opening of the anastomosis.

Fig. 10 (Experiment 8, Dog L4).—Blood pressure readings: upper, a definite drop in general blood pressure on opening the fistula at operation, Dec. 20, 1922; lower, no change on closing and opening the fistula five months later.

fall in blood pressure could be rectified by two procedures, the pressure being completely restored by closure of the proximal artery, and incompletely restored by closure of the vein proximal to the fistula (Fig. 11). The result of the second procedure was somewhat puzzling, but it was found repeatedly, and is in accord with our explanation that the cardiovascular changes attending an arteriovenous fistula are almost wholly the direct result of a free return flow of arterial blood to the heart. That this free return flow causes a rise in venous pressure proximal to the fistula is evident from the following experiment:

EXPERIMENT 9.—The right common iliac vessels were exposed, and an anastomosis 2.5 cm. long was performed in the usual manner. A small bore

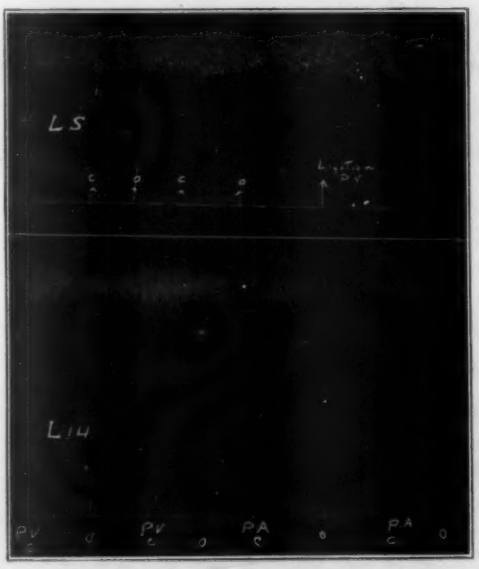


Fig. 11.—Blood pressure by readings: upper, Dog L5: marked effect on general pressure by the closing and opening of vein proximal to a fistula; permanent increase by ligation of the vein; lower, Dog L14: left femoral arterial pressures, dependent on conditions at right femoral fistula; proximal vein closed, pressure increased; proximal vein opened, pressure decreased; proximal artery closed, pressure increased; proximal artery opened, pressure decreased.

glass cannula was introduced into the vena cava through a small tributary vein 4 cm. above the anastomosis. When the fistula was opened, the general arterial pressure obtained in the left carotid artery dropped; there was an increase in pulse pressure, and a concomitant increase in vena cava pressure (Fig. 12). The changes in the pulse rate, in an already rapid heart, were not conspicuous, no doubt because of the surgical shock attending this necessarily prolonged experiment. A second glass cannula was introduced into the vena cava through the

Fig. 12 (Experiment 9).—Blood pressure readings: fall in carotid pressure and rise in vena cava pressure with the opening of fistula at *O;* increase in carotid pressure and fall in vena cava pressure with the closing of fistula at *C*.

Fig. 13 (Experiment 9).—Intimate relationship between carotid and vena cava pressure dependent on condition at the fistula: carotid pressure high and vena cava pressure low with fistula closed; carotid pressure low and vena cava pressure high with fistula open (vena cava pressures obtained in renal vein).

right renal vein, 10 cm. above the fistula. Opening and closing of the fistula resulted in alterations in arterial and venous pressure with perfect regularity, a fall in arterial pressure, and a rise in venous pressure when the fistula was opened with the reverse alterations when the fistula was closed (Fig. 13).

Corroborative observations were obtained in Dog L3.

EXPERIMENT 10.—March 10, four months after the formation of a common iliac fistula, Dog L3 was killed because of distemper. The fistula was exposed, and the left femoral artery and right renal vein cannulized for pressures. In spite of a blood pressure in the left femoral artery of only 70 mm. of mercury, the opening and closing of the fistula resulted in very evident blood pressure alterations; the arterial pressure increased on closure of the fistula, and the venous pressure (proximal to the fistula) lowered. The venous pressure in the renal vein was then determined directly and the results tabulated (Fig. 14). There was a constant difference of pressure of from 2 to 2.5 cm. water. This seems rather remarkable in view of an arterial pressure of only 70 mm. of mercury. The changes in the vena cava when the fistula was opened and closed were strikingly visible; it was tense, distended, and unusually large when the fistula was opened, becoming quite lax, easily compressible, and partially collapsed with the fistula closed by compression. There was no uncertainty in the

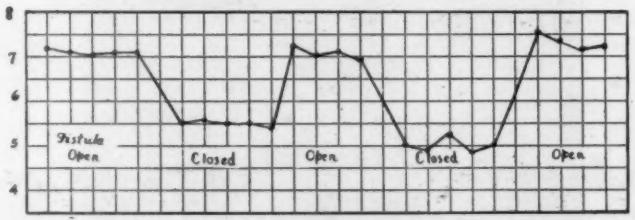


Fig. 14 (Experiment 10, Dog L3).—Alterations in vena cava pressure (renal vein) with the opening and closing of iliac fistula (venous pressure in centimeters of water in renal vein).

minds of four immediate observers that the opening of the fistula caused a marked increase in the pressure within the vena cava proximal to the fistula. The red oxygenated arterial blood could be seen coursing through the vena cava toward the heart at least 15 and 20 cm. above the fistula.

With such a graphic portrayal of the increased venous pressure in the presence of only 70 mm. of mercury pressure in the arteries, is it not fair to assume that even greater venous pressure would have been observed if the arterial pressure had been at its normal level of about 180 mm.? Additional corroborative observations were made in Dogs L26 and L28 (Fig. 15). In each case, closure of the fistula reduced the vena cava pressure and opening the fistula increased it.

Additional, almost irrefutable, evidence of this increased venous pressure is furnished by nature's adaptive response to its presence. It has been repeatedly demonstrated that the wall of the proximal vein in an arteriovenous aneurysm assumes the character of an artery—

"becomes arterialized." This has been noted by Reid,[4] by Vignolo,[5] and more recently by Paul Hermannes,[6] who describes the proximal vein in a fistula between the axillary vessels as gaping like an artery, with a thick and hard wall whose inner surface was smooth and uniformly rounded, so that macroscopically the vein gave the impression of being an artery. With reference to the central vein in a fistula between the subclavian vessels, cured by operation, he says that the vein lumen was larger than an aorta, with a thick and strong wall whose inner surface was smooth, and that the vessel gaped like an artery, so that macroscopically it was not distinguishable from the latter. He briefly summarizes the changes in the following paragraph:

The veins in arteriovenous aneurysm are changed in several ways as compared to their original condition, the more so, the longer the existence of the fistula.

Fig. 15 (Dog L26).—Rise in arterial pressure and fall in venous pressure on closure of iliac fistula (in spite of very low arterial pressure of 80 systolic).

The lumen is enlarged, the intima is considerably thickened, sometimes to within half of the entire vessel wall: in it lies a rich new production of elastic fibers, in which one can recognize a predominantly circular arrangement. The media is correspondingly thickened; besides a large amount of connective tissue which on the intima side is more finely fibered and contains numerous finest elastic fibers, there develops in it a rich ring of circular interlacing muscle fibers. The transition into adventitia is not certainly recognized, and our observations

4. Reid, M. R.: The Effect of Arteriovenous Fistula Upon the Heart and Blood Vessels, Bull. Johns Hopkins Hosp. **31**:43-50 (Feb.) 1920.

5. Vignolo, Quinto: Un Contributo Sperimentale all 'Anatomia e fisiopatologia dell' Aneurisma Arterio Venoso, Il Policlinico, Roma **60**:197, 1902.

6. Hermannes, Paul: Zur Frage der Arterialisierten Venen beim arteriovenosen Aneurysma, Brun's Beitr. z. klin. Chir. **130**:40-59, 1923.

concerning it are therefore also uncertain. However, one sees here besides thick, old, stretched elastic fibers, also a number of finest fibers, which one must consider as newly developed.

Carrel [7] has described similar changes occurring in a segment of vein transplanted in an artery, the changes being explained on the purely mechanical basis of increased pressure.

Eisenbrey [8] reports a most remarkable change in the vessels above a femoral arteriovenous fistula, these changes occurring only in that portion of the circulatory system represented by heart-artery-fistula vein, through which, according to our theory, a large volume of blood, short-circuited by the fistula, was constantly passing. Eisenbrey says (italics ours):

The heart was dilated and hypertrophied, the muscle wall pale and flabby, the right auricle thickened and greatly dilated. Mitral and tricuspid orifice slightly thickened, but not distorted, and without vegetation. The aortic orifice was dilated, the leaflets sclerosed, but not ulcerated, nor distorted. The arch of the aorta was dilated, and showed advanced atheromatous changes. The aorta from arch to bifurcation showed early atheromatous changes. *The right common iliac artery presented a relatively normal appearance,* while on the left side, the artery had undergone most pronounced changes. . . . The adjoining orifices in artery and vein had become closely and densely adherent to each other, and a smooth surfaced opening of irregular rounded outline about 0.7 cm. in diameter persisted. . . . . For a distance of 1.5 cm. distal to the point of communication the artery was dilated and the wall markedly sclerosed, and atheromatous; *below this point it appeared normal.* The vein wall was thickened and dilated for a distance of 3 cm. below the communication, where a pair of well developed valves was found. *Below these valves the vein appeared normal.* Above the communication both artery and vein were dilated to about 2.5 cm. in diameter. The arterial wall was sclerosed and atheromatous and contained calcareous plaques. The vein wall was considerably thickened. In a modified degree the vein followed the tortuosity of the artery and the *dilatation and thickening of the wall extended into the inferior vena cava while the right common iliac vein appeared normal.* Except for sclerosis and marked atheromatous change about its mouth the internal iliac artery appeared normal.

From the condition of the vena cava, its dilatation and thickening, it is reasonable to suppose that the pressure within it had been increased over a long period of time and the findings in the heart, especially on the right side, may well have been due to the distal arteriovenous communication.

It seems most significant that the sclerotic changes in this case were wholly limited to that portion of the circulatory system represented by the heart-artery-fistula vein circuit, through which a large volume of blood was constantly passing.

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7. Carrel: Transplantation biterminale complete d'un segment de veine sur une artere, Compt. rend. Soc. de biol. **59**:412-413, 1905.

8. Eisenbrey, A. B.: Arteriovenous Aneurysm of the Superficial Femoral Vessels, J. A. M. A. **61**:2155-2157 (Dec. 13) 1913.

The repeated observations both clinical and experimental of an arterialization of the vein proximal to an arteriovenous fistula seem to give almost incontrovertible evidence of an increased pressure in that portion of the venous system proximal to the fistula and in the vicinity of the fistula. It is evident experimentally that a well functioning, healthy heart muscle will react immediately to such an increased venous pressure by more rapid action, just as it happened in the work of Bainbridge. With a well functioning heart muscle there need not necessarily be any marked increase of pressure in the peripheral venous system or even a markedly enlarged liver, such as occurs in the presence of a dilating diseased heart, which produces an increased venous pressure by failure to propel forward the accumulating blood. Certainly, such a situation is not comparable with that found in an arteriovenous fistula in which, at least for a long period, there is no question of a failing heart. On the contrary, as experiments in Part III will show, there is a very decided augmentation of the vigor and rapidity of heart action as the result of the more than adequate inflow into the heart in the presence of a fistula.

## SUMMARY

The opening of an arteriovenous fistula causes an acceleration of the pulse rate, and a fall in blood pressure. Closure of a fistula retards the pulse rate and increases blood pressure. These latter changes are slight at first but gradually increase; they are dependent on the dilatation of the vessels leading to the fistula, which in turn is dependent on the size of the fistula and the amount of blood short-circuited through the fistula. Small fistulas may show regressive changes; larger fistulas produce progressive changes in retardation of pulse and increased blood pressure, the progress of the changes developing, so we presume, *pari passu* with the increasing blood volume, demonstrated in Part I, which in turn produces a dilatation of the heart-artery-fistula vein system.

The fall in blood pressure on opening an arteriovenous fistula affects both systolic and diastolic levels. The compensatory adjustment following the introduction of a fistula, mainly an increasing blood volume, result in a complete recovery of systolic pressure, sometimes in excess of that present before the production of the fistula. There is, however, only a slight recovery of the diastolic level and this remains permanently lowered in the presence of a fistula, thus producing a very distinct increase in pulse pressure.

Excision of the fistula results in a temporary excessive increase in both systolic and diastolic levels, with a gradual adjustment to normal levels, and a distinct decrease in pulse pressure to a normal figure, these changes being due to a gradually decreasing total blood volume.

There is an increase in venous pressure in the vein proximal to an arteriovenous fistula. This increased venous pressure need not necessarily produce an increase in peripheral venous pressure in the presence of a well functioning, unimpaired heart muscle, which reacts to this increased venous filling by an acceleration of heart action. In the presence of a large fistula between large vessels, there is a permanent increase in pulse rate to take care of the increased venous filling.

# EXPERIMENTAL STUDIES IN ARTERIOVENOUS FISTULAS

## III. CARDIAC DILATATION AND BLOOD VESSEL CHANGES *

EMILE HOLMAN, M.D.
Resident Surgeon, Peter Bent Brigham Hospital
BOSTON

A frequent, though by no means invariable, phenomenon observed clinically in arteriovenous fistulas has been the increase in the size of the heart, associated with an increase in the size of the artery and vein proximal to the fistula. Since William Hunter's first description of a proximal dilatation of the artery in a case of arteriovenous aneurysm, this phenomenon has puzzled physicians in their efforts to develop an adequate explanation for its occurrence in certain instances and its absence in others. Most writers, unwilling to accept a purely mechanical explanation of the changes, attribute them to a vital factor comparable to that which causes the vessels of a gravid uterus or of a growing tumor to dilate. Others have attributed the proximal dilatation to a lessened arterial tension, a disuse atrophy. Few explanations of the cardiac enlargement are available. Lewis and Drury [1] explain it on the basis of a deficient nutrition of the heart muscle, referring to the dilatation of the artery as an attempt to compensate for a deficient blood flow to the part beyond the fistula. According to this theory, a decreased flow causes the cardiac dilatation, and an increased flow, the arterial dilatation. Experimentally, Reid [2] was able to produce an increase in heart shadow and cardiac hypertrophy in one dog in which a fistula between the carotid artery and external jugular vein had been made, the animal surviving thirty-three months. In his discussion, Reid stresses the long duration of the fistula as being the important factor in producing these changes. Obviously, however, duration, while it may determine the extent of the changes, cannot explain the mechanism of their appearance. The following experimental observations are presented as evidence that duration is a minor factor, that dilatation may occur very rapidly, depending on the size of the fistula, its location, and particularly on the amount of return flow to the heart. The very intimate relationship that was found to exist between the size of the fistula and the blood pressure and blood

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* From the Laboratory for Surgical Research, Harvard Medical School, and the Hunterian Laboratory of Experimental Surgery, Johns Hopkins Medical School.

1. Lewis, T., and Drury, A. N.: Observations Relating to Arteriovenous Aneurysm, Heart **10**:301-389 (Oct.) 1923.

2. Reid, M. R.: The Effect of Arteriovenous Fistula Upon the Heart and Blood Vessels, Bull. Johns Hopkins Hosp. **31**:43-50 (Feb.) 1920.

volume variations (Parts I and II) is also apparent in a study of the visceral changes. This purely mechanical factor in the production of changes incident to a fistula is intimately associated with the known hydraulics of the circulatory system, on which depends the maintenance of an adequate blood pressure. When approached from this physiologic point of view, the changes are seen to occur as a natural sequence of events, and are easily understood.

## EXPERIMENTAL DATA

*Effect of Small Fistulas.*—Dog L2, with a comparatively small fistula (Fig. 1) accompanied by almost no demonstrable blood pressure or blood volume changes, showed no dilatation of the proximal vessels or cardiac enlargement after five months (Fig. 2).

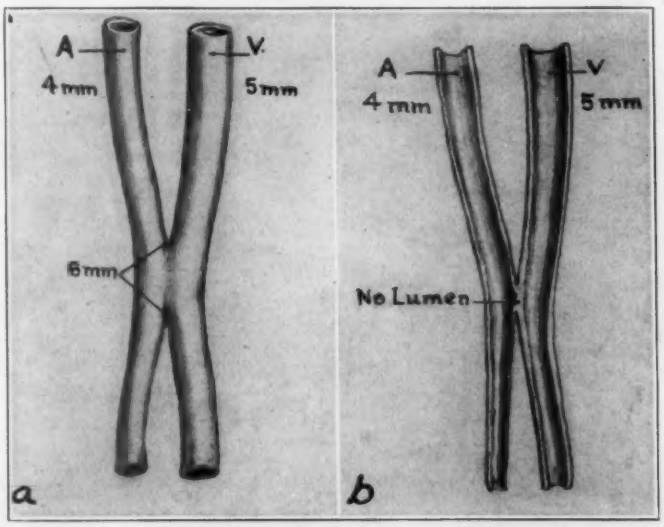


Fig. 1 (Dog L2).—A small fistula between common femoral vessels: *a*, established Jan. 12, 1923; *b*, no change revealed in size of proximal vessels and a complete healing of the fistula at necropsy, June 13. The thrill was felt for the last time about seven weeks before the necropsy.

Dog L4, also with a small fistula, showing occasional transitory changes in pulse rate on the closing of the fistula, with a slight increase in total blood volume, exhibited at necropsy a slight visible dilatation of the proximal vein, but no visible increase in the size of the proximal artery. The vessels were carefully dissected free, injected with sodium iodid solution, and a roentgen-ray photograph was taken. Examination of this photograph revealed, as before, a visible dilatation of the proximal vein, in addition to a detectable increase in the caliber of the proximal artery (Fig. 3). There was a slight dilatation of the ventricular cavities without much cardiac enlargement. One may infer from these observations that the first visible change to occur is in the vein, followed by subsequent visible alterations in the artery and heart, if they occur.

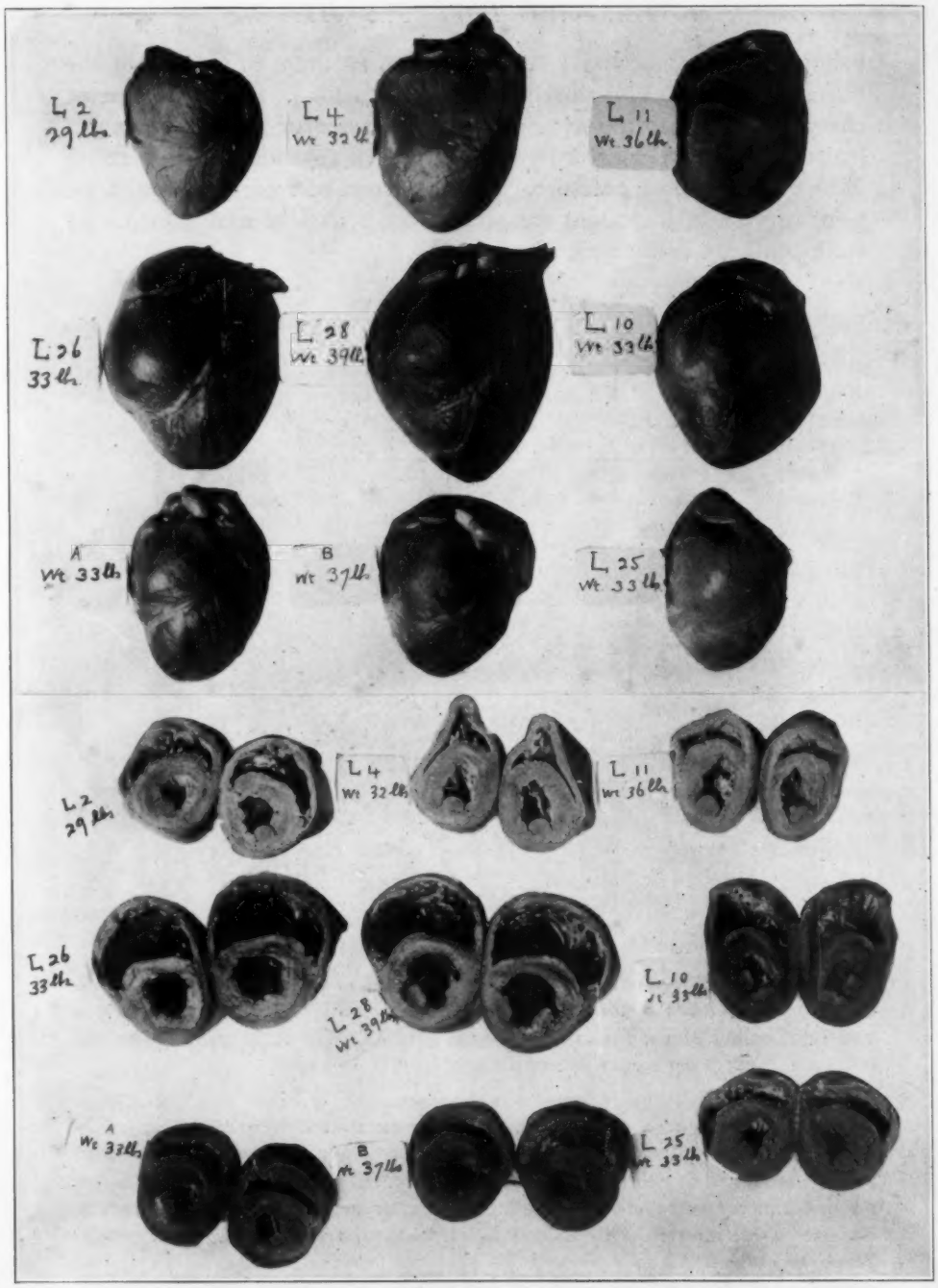


Fig. 2.—Hearts removed at necropsy: the upper group illustrates the marked enlargement of L26, L28, and to a lesser degree of L10; L2, L4 and L11 showed no enlargement. A and B are hearts taken from normal animals of practically the same build and size as L26 and L28, the hearts having been obtained on the same days and treated in an identical manner. The lower group shows that the enlargement is conspicuously a dilatation of the ventricular cavities.

Fig. 3 (Dog L4).—Suggestive dilatation of very slight degree of right femoral artery and vein proximal to a small femoral fistula of six months' duration; vessels injected with sodium iodid.

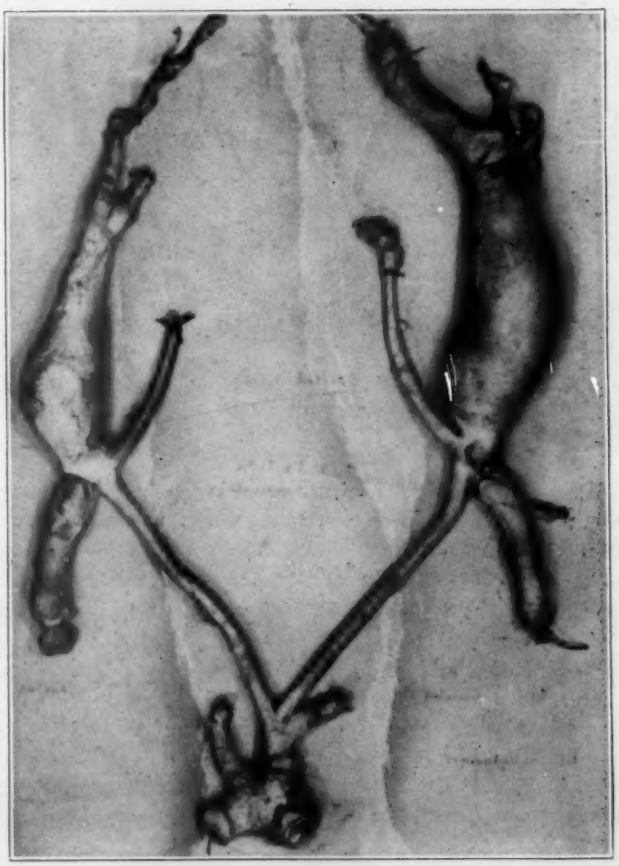

Fig. 4 (Dog X10).—Specimen showing no dilatation of vessels central to fistula between jugular and carotid vessels.

Dog X10, an interesting specimen, was obtained several years ago, when small fistulas about 4 to 6 mm. long were being established between the carotid and jugular vessels on both sides of the neck. The characteristic thrill was present until the dog was killed twenty-five months later. Dissection of the vessels revealed no dilatation of the proximal vessels, but there was marked ampullar dilatation of the vein distal to the opening (Figs. 4 and 5). Careful examination of the two specimens disclosed a definite obstruction to a free return flow to the heart; this undoubtedly accounted for the absence of dilatation of the proximal vessels, in spite of the long duration of the fistula.

A similar observation is recorded by Hoover and Beams.[3] Both carotid arteries of a dog were turned into the adjacent external jugular veins with the

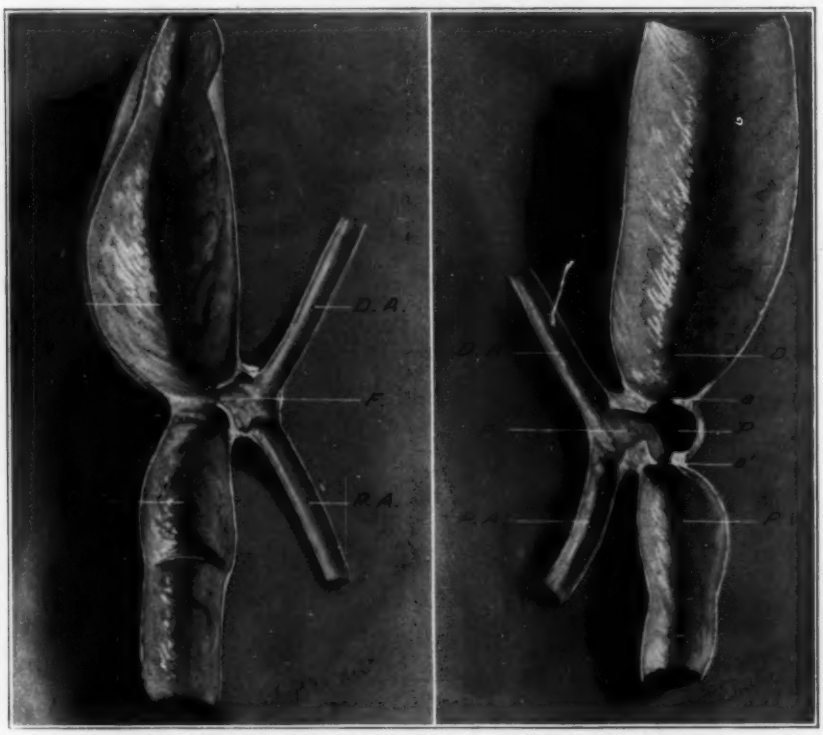


Fig. 5 (Dog X10).—Cross sections, revealing only small communications present, with definite obstruction to venous return to heart. PA: proximal artery; PV: proximal vein.

confident expectation that the heart would dilate. Much to their disappointment, at the end of six months there was no appreciable alteration in the size of the heart. The failure of the heart to dilate is unquestionably attributable to the insufficient return flow to the heart, due to the size of the fistula in each case, as each was probably smaller in diameter than that of the feeding artery.

3. Hoover, C. F., and Beams, A. J.: The Diagnosis and Pathologic Physiology of Arteriovenous Aneurysm, Arch. Int. Med. **33**:1-22 (Jan. 15) 1924.

In Dog L11, a large fistula 3.5 cm. long, was established between the femoral vessels, but the vein was ligated proximal to the fistula, thus preventing any return flow to the heart (Fig. 6). At necropsy three and one-half months later, there was no alteration whatever in the size of the proximal artery or vein, and no change in the size of the heart (Fig. 2).

*Changes Accompanying Large Fistulas.*—In Dog L10, with a large femoral fistula, 3.1 cm. in length, definite dilatation of the proximal vessels was noted three and one-half months after the formation of the fistula (Fig. 7). There was also an evident enlargement of the heart (Fig. 2).

Dog L14, weighing 13.6 kg., was operated on March 24, 1923, and a fistula 3.5 cm. long established between the right femoral vessels measuring 3 and 5 mm., respectively. The dog was found dead forty-five days later, following a fight in which he received numerous puncture wounds of the abdomen. The vessels

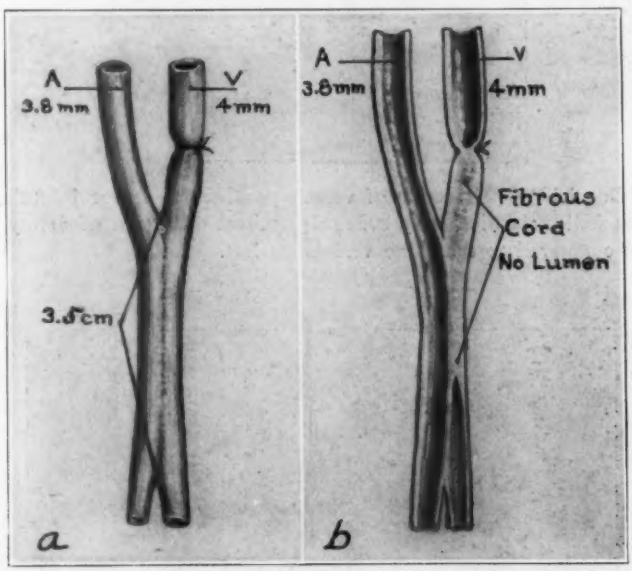


Fig. 6 (Dog. L11).—Large femoral fistula, with proximal vein ligated, thus preventing the return flow to heart: *a*, established March 2, 1923; *b*, no dilatation of proximal vessels revealed at necropsy, June 16, 1923. Anastomosis healed.

proximal to the fistula had increased in size to 4 and 6 mm., respectively, and the heart was considerably enlarged (Fig. 8).

Nov. 29, 1922, a femoral fistula 3.4 cm. long was made in Dog L3 between vessels 4 and 6 mm. in diameter (Fig. 9 *a*). Repeated examinations (Part II) yielded very striking blood pressure changes on the opening and closing of the fistula. Very definite blood volume alterations were also obtained (Part I). On June 15, 1923, the fistula in the femoral region was excised; the vessels proximal to the fistula exhibited an evident dilatation to 7 and 8 mm., respectively (Fig. 9 *b*). The heart also had undergone a very marked dilatation in six and one-half months' time (Fig. 10). Following excision of the fistula, blood volume and blood pressure adjustments, as well as cardiac contraction, took place (Fig. 11 *c*). It is instructive to compare the size of the heart on June 23 with that on July 18,

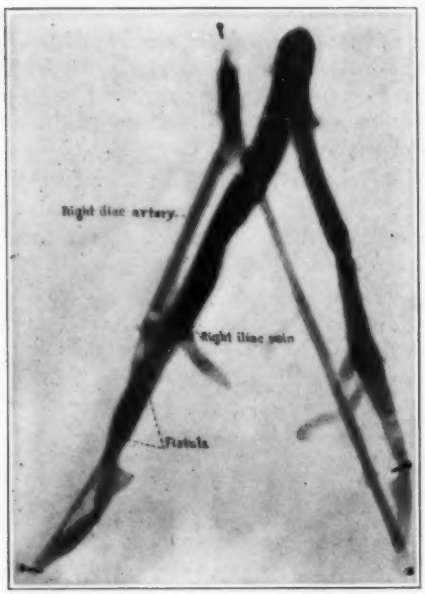


Fig. 7 (Dog L10).—Dissection of vessels proximal to femoral fistula, injected with sodium iodid, showing very evident proximal dilatation of artery and vein on right as compared to the same vessels on left.

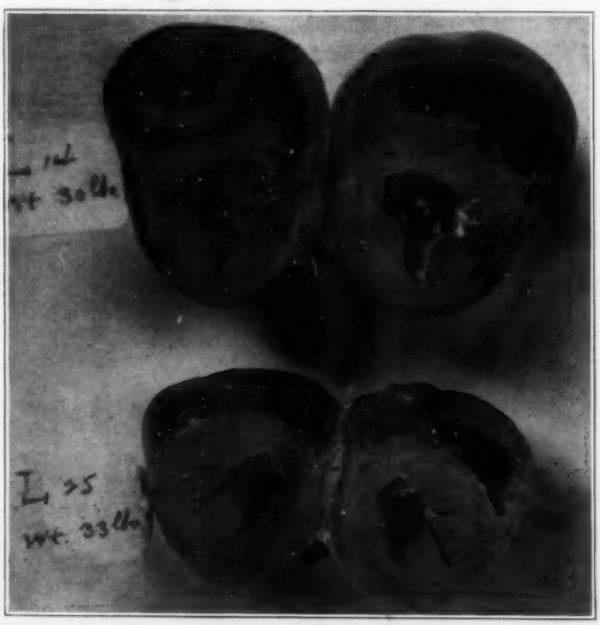

Fig. 8 (Dog L14).—Marked increase in cardiac area, with dilatation of ventricles forty-five days after formation of fistula, as compared with normal heart of similar sized animal, L25.

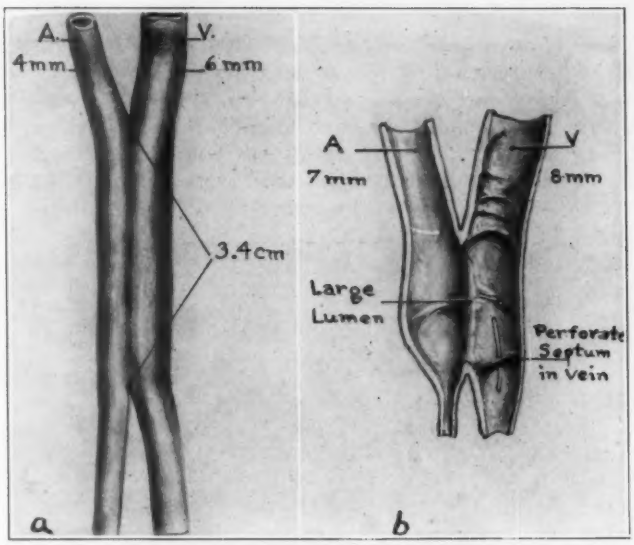


Fig. 9 (Dog L3).—Large femoral anastomosis: *a*, established Nov. 29, 1922; *b*, fistula excised June 15, 1923, showed proximal dilatation of vessels.

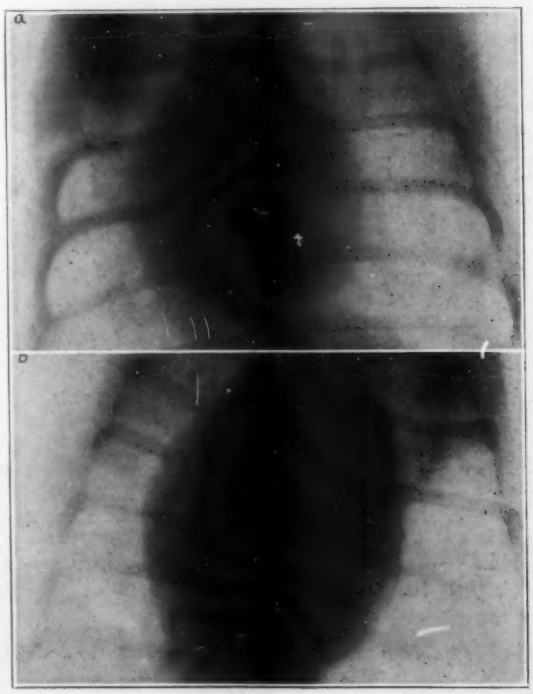


Fig. 10 (Dog. L3).—Cardiac shadow: *a*, Nov. 28, 1922 (femoral fistula established November 29); *b*, cardiac shadow May 8, 1923.

bearing in mind that a blood volume determination of 2,150 c.c. was made on June 23 and 1,510 c.c. on July 18. A decreasing heart shadow was evidently dependent on a decreasing blood volume. Nov. 9, 1923, a second anastomosis between the right, common iliac vessels was established. In June, at the excision of the femoral fistula, the femoral artery was noted as having a diameter of 7 mm. At the operation in November, the common iliac on the right had a

Fig. 11 (Dog L3).—Cardiac shadow: *a*, June 13, 1923 (fistula excised June 15); *b*, June 23; *c*, July 18.

diameter of only 5 mm., suggesting a regressive contraction of the vessel following excision of the femoral fistula. Following the formation of the iliac fistula, important changes again occurred in the total blood volume and in the size of the cardiac shadow (Fig. 12). In February, the animal began to fail gradually and was killed March 10, 1924, in a state of marked emaciation and of advanced

bronchial sepsis, due to distemper. The vessels were dissected free, and most instructive information was obtained (Fig. 13). The artery proximal to the fistula was dilated to almost twice the size of the left iliac artery, which was three and one-half mm. in diameter. The artery distal to the fistula was small and narrowed to approximately 3 mm., although at the operation for the removal of the femoral fistula performed June 16, this vessel was dilated to 7 mm.

It is obvious that regressive changes had occurred in the femoral and iliac arteries following excision of the femoral fistula just as regressive changes had occurred in the heart. However, with the production of the iliac fistula, a dilatation of the proximal artery had again recurred. We have, therefore, very positive evidence in this case that *a blood vessel will place itself in harmony with the amount of blood traversing it.* The heart at necropsy was definitely

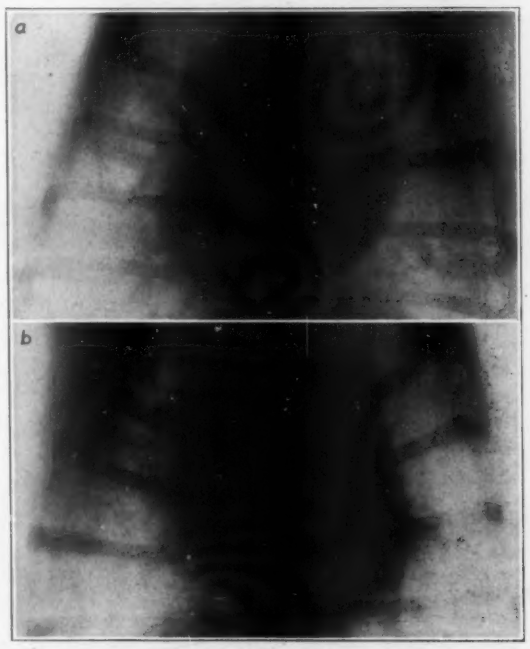


Fig. 12 (Dog L3).—Cardiac shadow: *a*, Oct. 31, 1923 (iliac fistula established November 9); *b*, December 4, showing a definite enlargement.

enlarged (weight 132 gm.), but not as conspicuously dilated as one might expect, apparently because of the marked emaciation and anemia accompanying the pulmonary sepsis. The liver was rather more firm than normal, but not obviously enlarged.

*Rapid Changes Due to an Aorta-Vena Cava Fistula.*—The following experiment performed on Dog L19 was most enlightening. Instead of establishing a fistula between the femoral or iliac vessels, it was decided to anastomose the abdominal aorta and vena cava just above the trifurcation. It is most important in this region not to make the fistula too large, for the immediate result may be complete paralysis of the lower limbs, and more remotely, that is, within several days, death from as yet undetermined causes, probably cardiac exhaustion in the

presence of a greatly reduced blood pressure. It is also important not to ligate the posterior branch of the aorta leading to the lower segment of the spinal cord, otherwise a complete paralysis of the lower limbs will follow, due presumably to an anemia of the cord. In three of the animals in which this procedure was attempted, L16, L20 and L22, there was complete paralysis of the lower extremities in two, and all the dogs died within seventy-two hours. No definite cause of death was ascertained in these three cases, the suture line was well healed, and no bleeding into the abdominal cavity had occurred in any case.

In L19, we were fortunate enough to make a fistula in this region compatible with life. On May 9, 1923, a fistula 1.2 cm. long was established between the aorta and vena cava just above the trifurcation (Fig. 14). On the day preceding the operation a roentgenogram of the heart had been taken (Fig. 15), and a pulse of 110 was recorded. During the operation, the pulse rose to 150. Imme-

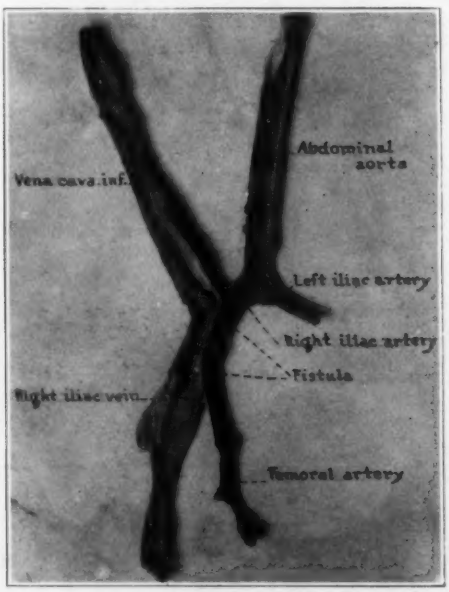


Fig. 13 (Dog L3).—Specimen removed Jan. 25, 1924, illustrating: (1) dilatation of right iliac artery proximal to common iliac fistula; (2) ampullar dilatation of right iliac vein opposite the fistula, and (3) the small normal sized right femoral artery beyond the iliac fistula (a femoral fistula was excised on the right on June 15, 1923).

diately after release of the clamps the pulse rose to 200, and at the end of the operation it had settled down to 170. The thrill was intense, extending well down into each thigh along the vessels, and a loud bruit could be heard as far as 45 cm. from the vessels. Both bruit and thrill were eliminated by closure of the proximal vein. There was a good vigorous pulse palpated in both femoral arteries at the conclusion of the operation, indicating that only a portion of the arterial blood stream was being diverted into the venous channels. There was no paralysis nor edema of the extremities following the operation, and the wound healed by first intention. About five days after the operation, a slight edema of

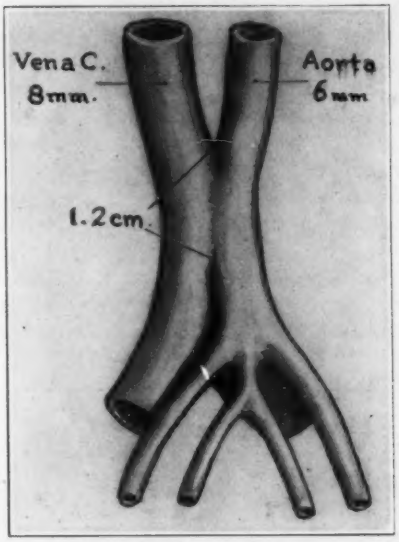


Fig. 14 (Dog L19).—Fistula between aorta and vena cava, established on May 9, 1923.

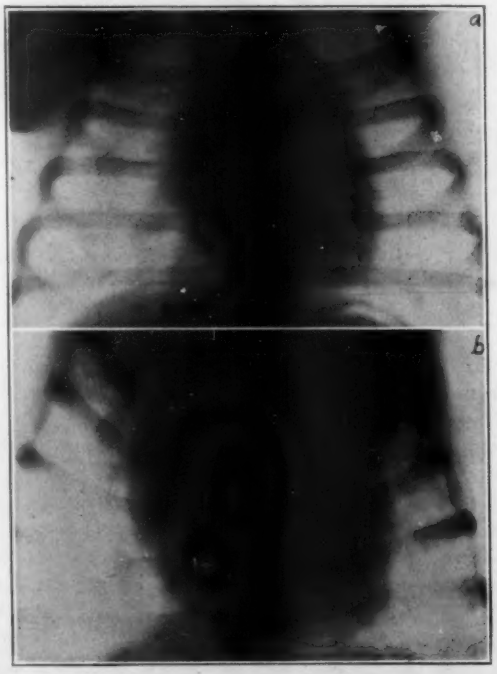


Fig. 15 (Dog L19).—Cardiac shadow: *a*, May 8, 1923 (aorta-vena cava fistula established May 9); *b*, May 25.

the two lower breasts appeared, which gradually increased until it involved the lower abdominal wall. Very large, tortuous veins appeared on the surface of the abdominal wall and filled from below. The heart during the first few days after the operation maintained a rate of from 160 to 180, and could be felt beating rather feebly against the chest wall. Then, there occurred, however, a gradually increasing vigor of heart action, and during the next two weeks the heart could be felt over the entire thoracic cage both front and back, beating with extraordinary vigor, with a rate constantly around 180. Blood pressures were not obtainable with our cuff, as the fistula lay above the femoral vessels, and it was considered unwise to sacrifice arteries elsewhere for cannulization. The dog during these three weeks remained very quiet in her cage, undue exertion producing dyspnea and, apparently, fatigue. In spite of the deep-seated location of the fistula, a thrill could be felt over the entire abdominal wall, with a loud bruit on auscultation. The increasing edema of the abdominal wall, a

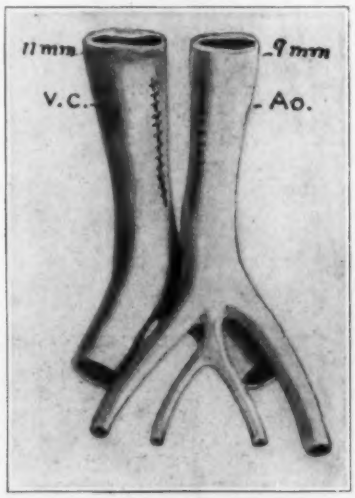


Fig. 16 (Dog L19).—Fistulous opening repaired June 1, 1923; dilatation of proximal artery and vein, twenty-two days after the formation of fistula may be noted.

gradual increase in the area of visible and palpable pulsation over the thoracic cage, together with the roentgen-ray evidence of a markedly dilated heart (Fig. 15), all indicated a probable, early decompensation unless the fistula was repaired. Accordingly, on June 1, twenty-two days after the formation of the fistula, the dog was again anesthetized, and the fistula repaired by separating the aorta and vena cava, and closing the longitudinal slits in the two vessels (Fig. 16). The abdomen contained a large amount of clear, straw-colored fluid, and, although on palpation the liver felt large, there was no ascertainable pulsation in it. Following the closure of the fistula, a definite pulse could be felt in both femoral vessels, thus insuring the reestablishment of the normal circulating relations. The pulse rate at the end of the four hour operation was 192, and the dog was in a state of considerable shock. The operation was a tedious and difficult one because of the presence of fibrous deposits around the site of the recent operation. The following day, however, the rate had dropped to 144, and

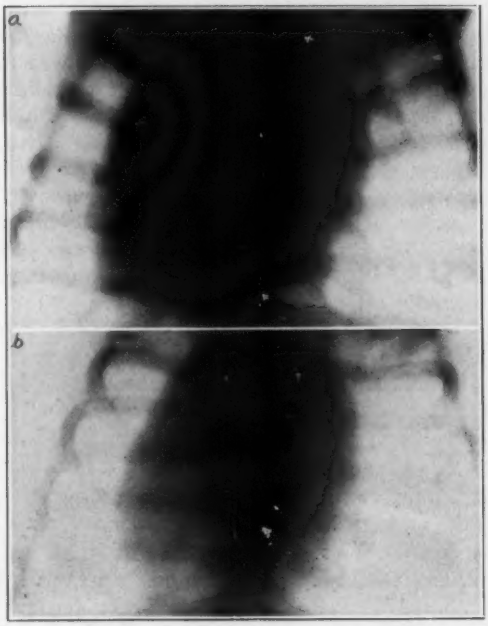


Fig. 17 (Dog L19).—Cardiac shadow: *a*, May 30, 1923 (fistula repaired June 1); *b*, three days after the repair of the fistula.

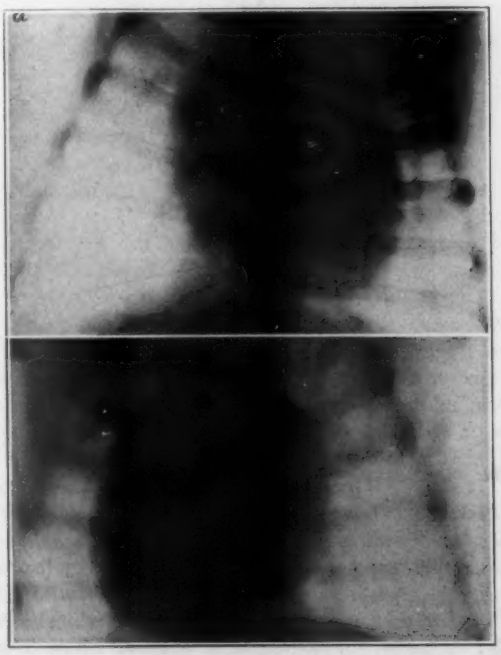


Fig. 18 (Dog L19).—Cardiac shadow: *a*, July 18, 1923; *b*, September 18.

the heart action gradually subsided in vigor until, three days after the repair, it could be felt, as normally, only over the anterior portion of the chest wall; the rate had again fallen to 126 for the first time since the initial operation. The edema of the abdominal wall gradually subsided, and within two weeks the quiet, easily fatigued dog had been transformed into an active, vigorously barking animal, exhibiting marked interest in her surroundings.

The roentgen-ray evidence obtained here was most convincing as a demonstration of the causal relationship existing between cardiac dilatation and an arteriovenous fistula. That it is mostly dilatation is apparent from the rapid increase in the size of the cardiac area following the formation of a fistula, and its rapid decrease within three days after the closure of the fistula (Fig. 17). The most recent roentgenogram, however, shows still some increase in cardiac shadow (Fig. 18), indicating that some hypertrophy must have developed during the three weeks of excessive heart action. It should be noted that there was definite increase in the caliber of the aorta and vena cava proximal to the fistula at the time of the second operation (Fig. 16).

*Cardiac Dilatation Immediately Following Production of a Fistula.*—The following fluoroscopic observations were made on Dog L22. A fistula was produced between the aorta and vena cava about 1.2 cm. long. Before the clamps were released, the dog was placed on the fluoroscopic table and the heart outlined. In the dark, the four clamps were supposedly removed, with the expectation that the heart would dilate. To our amazement, however, it decreased in size, and there was no thrill present. Investigation revealed a clamp still occluding the vein proximal to the fistula, the removal of which immediately caused the heart to increase in size. Closure of the proximal artery caused a decrease in the heart shadow equal to that noted before the removal of any of the clamps. Closure of the proximal vein, however, with the proximal artery open, caused an even greater decrease in cardiac area.

Confirmatory evidence of a similar nature was obtained in the following experiment:

In Dog L29, a fistula 1.2 cm. long was made between the aorta and the vena cava. The pulse rate before the fistula was opened was 132. One minute after the opening of the fistula, it had risen to 148, and five minutes later to 180. Closing the proximal vein reduced the pulse to 156; reopening it accelerated the pulse to 184. At the end of the operation, the pulse rate was 210, and it continued at this high rate until the death of the animal six days later. The striking observations in this case, however, are shown in Figure 19. All plates were taken at the same distance without the slightest change in the animal's position. Figure 19 *a* shows the size of the heart before the opening of the fistula, with clamps on the abdominal aorta and the vena cava still in situ. The size of the heart in this plate agrees well with that obtained on the previous day. Figure 19 *b* shows a definite increase in cardiac area five minutes after the opening of the fistula. Clamping the proximal artery resulted in a return to the previous area (Fig. 19 *c*). Opening of the proximal artery and closure of the proximal vein produced a remarkable diminution in cardiac area as shown by Figure 19 *d*. In spite of this greatly decreased cardiac area, and in spite of the diminished venous return (or perhaps because of it!) the pulse rate was definitely retarded with the vein closed, as compared to the rate with the fistula opened. The decrease in cardiac area with the vein closed is obviously due to a filling up of the extremities distal to the fistula with arterial blood that cannot find its way back to the heart.

*Early Changes Due to Iliac Fistulas.*—The more rapid appearance of visceral changes in the presence of iliac fistulas as compared to femoral fistulas was illustrated in the case of Dog L26, a young collie, weighing 11.8 kg. On Aug. 29, 1923, an anastomosis 3 cm. long was established between the right common iliac vessels, the diameters measuring 4 and 6 mm., respectively. A pulse of 120 before operation rose immediately after the opening of the fistula to 168. On the following day the right leg and thigh were greatly swollen, and only a feeble pulse was palpable in the right femoral artery. In the left femoral artery could be felt a full vigorous pulse, which on auscultation gave the characteristic pistol

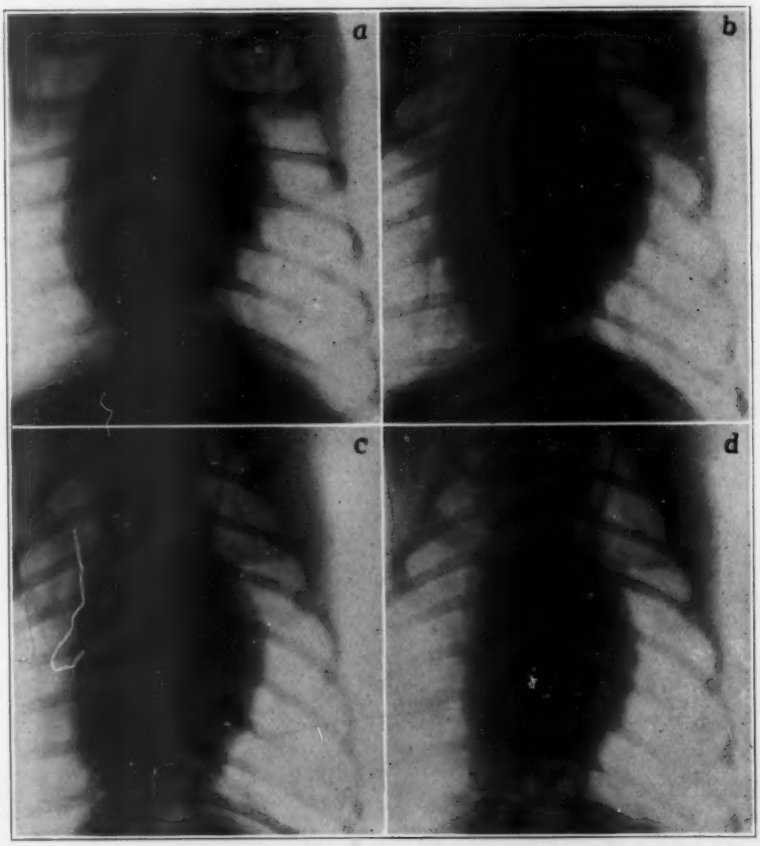


Fig. 19 (Dog L29).—Cardiac areas: *a*, fistula completed, clamps on aorta and vena cava still in situ; *b*, fistula open—heart shadow increased; *c*, proximal artery closed—cardiac area reduced; *d*, proximal artery opened, proximal vein closed, marked decrease in cardiac shadow, with heart pulsating more slowly than in *b*.

shot usually noted in the presence of aortic regurgitation. The right leg became very markedly swollen, and circulation was so much impaired that a large, dollar-sized area of dark, gangrenous slough appeared over the right knee, which did not heal for a number of weeks. Two months after the operation, the heart

could be felt beating rapidly and vigorously against the thoracic cage, the palpable and visible cardiac pulsation having greatly increased, as indicated also by the roentgen ray (Fig. 20). The blood volume also showed a gradual increase from 1,039 to 1,840 c.c., with an increase in weight from 11.8 to 15 kg. On Jan. 25, 1924, the dog was killed. The vessels proximal to the fistula were definitely enlarged, with a large ampullar dilatation of the vein opposite the fistulous opening (Fig. 21). The arterial blood could be seen swirling back through the vena cava from the fistula, arterial blood being noted by its color at least 15 cm. above the anastomosis. The heart as it lay in its bed was tremendously enlarged, appearing to be almost equal in size to the human heart. The right auricle and ventricle appeared particularly prominent. In comparison with another heart removed on the same day from a similarly built, long limbed animal, 15 kg. in

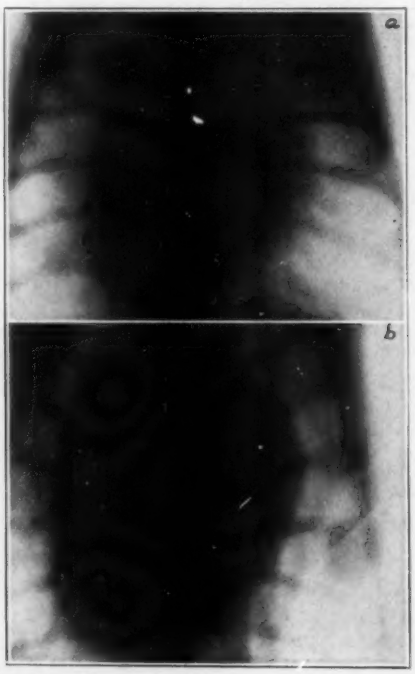


Fig. 20 (Dog L26).—Cardiac shadow: *a*, Aug. 20, 1923 (iliac fistula established August 29); *b*, Jan. 12, 1924.

weight, it appeared about twice the normal size (Fig. 2, upper). On section the marked dilatation of both ventricular cavities was at once apparent, the right ventricle showing this dilatation much more conspicuously (Fig. 2, lower).

Oct. 22, 1923, on Dog L 28, a large, female airedale, weighing 18 kg., an anastomosis between the right, common iliac vessels was made, the fistula being about 2.5 cm. long, and the vessels 5 and 7 mm. in diameter. When the clamps were released there was a marked acceleration of the pulse from 140 to 210. A loud audible bruit was present, obliterated completely by closure of the proximal vein. The pulse at the end of the operation was 210. The following day there was marked swelling of the right leg, with unusual prominence of the

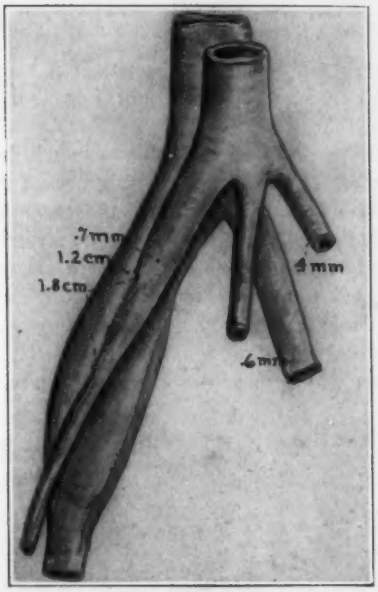


Fig. 21 (Dog L26).—Fistula between common iliac vessels established Aug. 29, 1923; necropsy Jan. 25, 1924, revealed proximal dilatation of artery and vein.

Fig. 22 (Dog L28).—Cardiac area: *a*, Oct. 20, 1923 (iliac fistula established October 29); *b*, December 4.

superficial veins. The measurements of the two legs a week later were: right, just above the ankle, 15 cm., left, 10 cm.; right, just above the foot, 31 cm., left, 25 cm.; right foot, 10.5 cm., left, 8.5 cm. By October 31, the swelling of the legs had begun to subside, disappearing completely within the three succeeding weeks. There was, however, a marked prominence of the veins of the right thigh, as well as those of the right side of the abdominal wall which filled from below. The pulse on November 30 was recorded as 170, with a very vigorous, palpable cardiac impulse over the entire chest wall. The roentgen-ray examination revealed a marked increase in the cardiac area (Fig. 22).

Jan. 29, 1924, three months after the formation of the fistula, the dog was killed. There was a definite enlargement of the vessels proximal to the fistula (Fig. 23), and the heart was enormously enlarged; again, a marked prominence

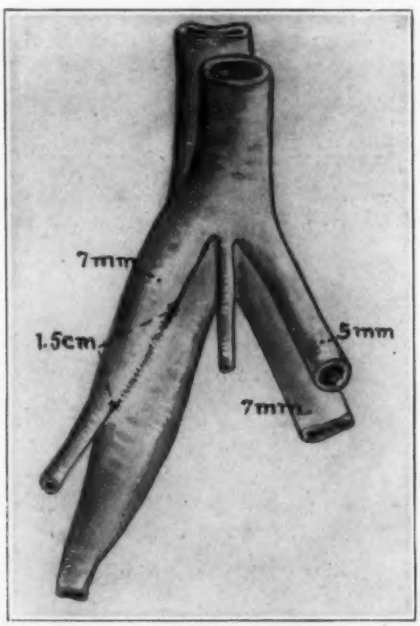


Fig. 23 (Dog L28).—Fistula between common iliac vessels established Oct. 29, 1923; necropsy, Jan. 29, 1924, showed dilatation of proximal artery and vein.

of the right side of the heart was present (Fig. 2, lower). It is also of interest to note that a microscopic examination of the liver and kidney in both Dog L26 and Dog L28 revealed no evidence whatever of chronic passive congestion. Both these animals possessed large fistulas, with increased vena cava pressure due to the fistula, but with rapidly and vigorously beating hearts.

The evidence seems conclusive that the development of the visceral changes depends on the location of the fistula, appearing more quickly and progressing more rapidly, the nearer to the heart the fistula lies, and the larger the vessels abnormally connected. Logically, this is what one would expect: the larger the vessel, the greater the capillary bed

short-circuited; hence, the greater the amount of peripheral resistance eliminated; and, necessarily, the greater the tendency for the blood to flow through the unobstructed fistulous opening back to the heart.

The swelling of the part beyond the fistula immediately following the formation of the fistula, and its gradual subsidence to complete disappearance is also of importance. The swelling is due to the flow of blood through the fistula into the distal venous bed. As the resistance here is developed by a gradual but complete filling of the circulatory bed, the blood is directed into a new channel, namely the vein proximal to the fistula. With the dilatation of this channel, practically the entire flow of short-circuited blood finds its way proximately instead of distally, and the swelling subsides.

*Proximal Dilatation of the Vessels: a Mechanical Phenomenon.*—An explanation of the proximal dilatation of the artery and vein is found by reverting again to our conception that the introduction of a fistula into the circulation results in establishing two systems of circulating blood where only one existed before, namely: A, heart-artery-capillary bed-vein, and B, heart-artery-fistula vein. It is obvious that the volume of blood that is short-circuited through system B depends on the size of the fistula, the character of the opening, and the absence of any venous obstruction proximal to the fistula. If the fistula is large enough, the entire volume of blood flowing into the artery proximal to the fistula will be diverted through the opening. If the fistulous opening is larger in cross-section than the artery, and if there is no venous obstruction proximal to the fistula, the only curb to the amount of blood that will flow through the fistula is, first, the limited distensibility of the arterial wall leading to it, and, second, the resistance to expansion of the artery offered by the tissues and structures surrounding the vessel. The artery, however, is an elastic tube and the surrounding structures are non-rigid, compressible tissues. As a result, resistance to distensibility is in fact small, and, obeying the maxim that flowing water seeks the path of least resistance, a larger and larger volume of blood will find its way into the vessel and through the fistulous opening, thus producing, by the force of the column of blood directed toward the fistula, a gradual distention and dilatation of the feeding artery and receiving vein. This distention responds slowly but certainly to the increasing blood volume flow, until a point is finally reached where the combined resistance to blood flow offered by the fistulous opening itself, plus the resistance offered by the limited distensibility of the vessel wall to a further dilatation of the vessel, equals the resistance to blood flow offered by the capillary bed in the system A. When this equalization of resistances is reached, the two systems are in equilibrium; the volume of blood flowing through the fistula will no longer increase, and therefore the total volume

will also remain constant. It is my belief that, when this equilibrium is reached, no further dilatation of the vessels and heart can or will occur. This dilatation is accomplished against the resistance to expansion offered by the vessel wall itself, and by the resistance offered by the surrounding tissues. It is, therefore, of necessity a slowly progressive process, and all writers have emphasized the long duration of the fistula as a prominent feature of the vessel and heart changes, when in reality the most important factor is the size of the fistula, and the absence of any obstruction in the vein proximal to the fistula. *Duration of the fistula is obviously not the reason for the dilatation, but in the presence of a large fistula it has a distinct bearing on the extent of the dilatation.*

Numerous clinical cases are recorded in which no detectable enlargement of the heart or blood vessels occurred, and from our experimental evidence we must assume that the fistulas in these cases were large

TABLE 1.—*Cardiac Hypertrophy in Arteriovenous Fistula*

| Number of Dog | Size of Fistula | Duration of Fistula, Days | Weight of Dog, Kg. | Weight of Heart, Gm. |
|---|---|---|---|---|
| L2 | Small femoral | 153 | 13.2 | 94 |
| L3 | Large iliac | 123 | Normally 15 kg.; at death 8 kg. | 132 |
| L4 | Small femoral | 178 | 14.6 | 103.5 |
| L10 | Large femoral | 103 | 15 | 130.5 |
| L11 | Large femoral (proximal vein tied) | 101 | 16.3 | 106 |
| L14 | Large femoral | 45 | 13.6 | 144 |
| L26 | Large iliac | 149 | 15 | 147 |
| L28 | Large iliac | 99 | 18 | 164.5 |
| A normal | None | ... | 15 | 112 |
| B normal | None | ... | 17 | 116.5 |

enough to give the customary thrill and bruit characterizing the lesion, but not large enough to permit much leakage of arterial blood into the venous system. Most of these cases have undoubtedly reached a stationary stage, and were it not for the discomfort of the persistent thrill, these cases could be allowed to proceed without surgical intervention, without any fear of harmful effects from the fistula. In those cases, however, in which demonstrable visceral changes are present, such as cardiac enlargement or dilatation of the vessels, it is evident also from our experimental studies that these changes may still be progressive at the time the patient is first seen, and that the gradually increasing burden on the heart must be eliminated, or decompensation and death will ensue.

*Cardiac Dilatation and Hypertrophy.*—The gradually increasing flow of blood through the slowly dilating proximal artery and vein increases the volume of blood flowing through the heart. Just as the vessels place themselves in harmony with the volume of blood flowing through them, so the heart will dilate in response to this increased volume flow through its chambers. The experiments that revealed a proximal dilatation of

the vessels also produced a cardiac enlargement, due mostly to dilatation. However, to take care of this increasing blood volume flow, the heart must increase its muscular effort: this was shown clinically and experimentally by the markedly increased heart action in the presence of large fistulas. Theoretically, hypertrophy would be a natural sequence of this increased heart action, and again our experimental evidence has borne this out, as a study of the accompanying table will indicate.

Briefly, Table 1 shows that in Dog L2, with a small fistula, without changes in cardiac size and without blood vessel changes, the heart weighed 94 gm. In Dog L4, with a small fistula but with a very slight increase in the size of the proximal vessels, and a slight dilatation of the ventricular cavities, the heart weighed 103.5 gm. In Dog L11, with a large fistula but with the proximal vein tied, without blood vessel changes and without increased cardiac areas, the heart weighed 106 gm. In Dogs L3, L10, L14, L26, and L28, with large fistulas, with cardiac enlargement, and with dilatation of the proximal vessels, the weight of the hearts were 132, 130, 144, 147 and 164 gm. respectively. Two normal dogs, similarly built, weighing 15 and 17 kg. had hearts weighing 112 and 116 gm., respectively.

The evidence furnished by this table seems conclusive that there is not only a dilatation but also a definite increase in the weight of the hearts obtained from animals with large fistulas, an increased weight which must represent hypertrophy. The explanation offered by Lewis and Drury of a deficient nutrition to the heart muscle seems hardly applicable.

## GENERAL SUMMARY AND CONCLUSIONS

In briefly summarizing the experimental evidence presented in the three preceding papers, the following points are emphasized:

The production of an arteriovenous fistula establishes two systems of circulating blood: A, the heart-artery-capillary bed-vein system, and B, the heart-artery-fistula vein system. The short-circuiting of a volume of blood through system B, with its greatly reduced peripheral resistance, introduces a large factor of inefficiency into the circulatory system with particular reference to blood pressure. The variable factors concerned in the maintenance of blood pressure are:

1. Cardiac output.
2. Total volume of circulating blood.
3. Capacity of system.
4. Peripheral resistance.

The lowered peripheral resistance in the presence of a fistula is compensated by changes in the remaining three variable factors, and in this way only is an adequate blood pressure level maintained.

The volume of blood diverted and short-circuited through the lowered resistance of the fistula depends entirely on the size of the fistula, and the absence of any obstruction in the venous return flow to the heart proximal to the fistula. A small communication produces only slight changes, and these may not be detectable. With our present instruments of precision the slight alterations necessary to compensate for a small leak are not demonstrable, nor do they produce any visible visceral changes. A slightly larger fistula may result in changes, only some of which are demonstrable. A fistula larger in size than the feeding artery, or large enough to divert a considerable flow back to the heart, invariably produces demonstrable changes, both immediate and remote. That these changes to a greater or lesser degree occur in the presence of every fistula, no matter what the size, seems beyond question, the extent in each case depending entirely on the volume of blood diverted through the fistula.

The immediate changes due to the production of a fistula are:

1. A fall in general arterial blood pressure affecting both systolic and diastolic levels.

2. An increase in pulse rate.

3. An increase in venous pressure, proximal as well as distal to the fistula, which is slight or pronounced, according to the size of the fistula.

The remote effects following the production of a fistula are:

1. A permanent diversion of part of the blood volume from the circulatory system of heart-artery-capillary bed-vein into the second circulatory system of heart-artery-fistula vein.

2. A gradual increase in the amount of blood volume flowing through the new system, heart-artery-fistula vein, depending on the size of the fistula.

3. A gradually increasing vigor of heart action.

4. A gradually increasing total blood volume, proceeding *pari passu* with the amount of blood diverted through the fistula.

5. A gradual dilatation of the heart, and of the artery and vein proximal to the fistula, to accommodate this increasing flow of blood through the fistula.

6. An hypertrophy of the heart due to the increased work necessary to propel forward this increasing volume of blood flowing through it.

7. A gradual recovery from the lowered blood pressure noted immediately after the formation of the fistula, the systolic equal or higher, the diastolic definitely lower than that existing before the formation of the fistula, thus producing a greatly increased pulse pressure.

The closure of an arteriovenous fistula should theoretically produce the reverse of all these changes, and experimentally as well as clinically this has proved to be the case.

The immediate changes subsequent to the closure of a fistula by compression or by excision are:

1. A rise in the general arterial blood pressure to a high point for several beats with a gradual fall to a level above that which was present before the production of the fistula. This rise in pressure involves both diastolic and systolic levels.

2. A marked decrease in pulse rate, and a subsidence of the increased vigor of heart action.

3. A decrease in venous pressure proximal to the fistula.

These immediate changes are obtained not only by closure of the artery feeding the fistula, but also by closure of the vein leading from the fistula toward the heart.

The remote effects following repair or excision of a fistula are:

1. A gradual readjustment in the general blood pressure, the systolic falling to a level approximately the same as that before elimination of the fistula, the diastolic remaining at a higher level than that present with the fistula open. The result is a decreased pulse pressure as compared to that present with the fistula open.

2. A slow recovery to the normal rate of a pulse retarded by repair of the fistula.

3. A gradual decrease in total blood volume, the real cause of the gradual fall and adjustment in blood pressure noted above.

4. A gradual subsidence of the dilatation of the heart, and also of the proximal artery and vein, proceeding *pari passu* with the decreasing volume of blood flowing through them.

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